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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY  
ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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L. Jolley, Ontario Federation of Labour  
E. Warren, Asbestos Information Association  
of North America  
D. Starkman, Asbestos Victims of Ontario  
J. McNamee, Government of Ontario

180 Dundas Street  
Toronto, Ontario  
Tuesday,  
June 16, 1981

VOLUME IX



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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME IX

INDEX OF WITNESSES:

DR. HANS WEILL	Examination-in-chief	Page 3
	Cross-exam (Bazin)	Page 110
	Cross-exam (Jolley)	Page 118
	Cross-exam (Warren)	Page 130
	Cross-exam (Starkman)	Page 146
	Cross-exam (Ublansky)	Page 154
	Cross-exam (McNamee)	Page 159
	Examination	Page 163

INDEX OF EXHIBITS

EXHIBIT # 7	Dr. Weill's curriculum vitae, articles and brief	Page 9
EXHIBIT # 8	Dr. Weill's dose-response curves	Page 80
EXHIBIT # 9	Slide from Dr. Weill's Risk of Respiratory Malignancy chart	Page 83
EXHIBIT # 10	Slide from Simpson report	Page 100
EXHIBIT # 11	Dement's doctoral thesis paper	Page 105
EXHIBIT # 12	Dr. Weill's transparency on latency period	Page 107

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10:00 a.m.

THE FURTHER PROCEEDINGS OF THIS INQUIRY  
RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

MR. LASKIN: Doctor, Mr. McNamee, who, as you know acts for the government, would like to address a few words to the Commission.

DR. DUPRE: Please, Mr. McNamee.

MR. McNAMEE: Yes, Mr. Chairman, Commissioners, you recall that Dr. Finkelstein mentioned certain ongoing studies the other day, which he was involved in, and I believe Dr. Dupre asked where they were. Just to indicate that we weren't holding anything back, and I think that...I don't think anybody really thinks so...paragraphs 146 and 147 of our brief dated February, 1981, to the Royal Commission, located on page 61 of the brief, mentions these studies. I believe one is already being submitted to the Commission. It's called, Mortality Among Workers Receiving Compensation for Asbestosis in Ontario, Appendix Ten. I believe that's already in the Commission's hands.

Paragraph 147 states, "The branch has two major studies underway of asbestos-exposed workers at the Canadian Johns-Manville Company Limited, Scarborough Plant. Both are attempting to explore





MR. McNAMEE: (cont'd.) "exposure-response relationships, one by studying the mortality experience, and the other, the incidence of asbestosis in long-term employees."

It states, "The broad objective is to develop an exposure model which may have practical application in understanding the hazards of asbestos, and in the development of exposure limits".

Now, I've given Mr. Laskin four copies of the two studies prepared by Dr. Finkelstein, and one for Mr. Laskin to refer to the Commission.

The blue cover is, "Asbestos Among Long-Term Employees of an Ontario Asbestos Cement Factory", and some copies have previously been given to labour/management representatives... at least some of the labour/management representatives.

The second study is an unbound study headed, Draft, and it's called, "Mortality Among Long-Term Employees of an Ontario Asbestos Cement Factory". It's headed, Preliminary Report, and Dr. Murray Finkelstein explains that that has a special significance to people in the business. It means that it's part of an ongoing series of reports, not necessarily that data is inaccurate and subject to correction. It's sort of the first step in a series of reports.

This really deals with workers with nine years and more of exposure, and Dr. Finkelstein advises me that he also has an ongoing report that is not even in draft stage yet, of workers with less than nine years exposure. It will be several months before he completes that. It might be completed during the term of the hearings.

Dr. Finkelstein is prepared to be called as a witness by the Commission. He would like, though, if it be some time toward the latter part of July, so that he would have time to prepare for examination and cross-examination.





MR. McNAMEE: (cont'd.) That's my submission.

DR. DUPRE: Thank you, counsel.

5 Are there any parties with standing here today who do not have copies of what are now exhibits five and six?

Do you all have them? Mr. Warren, do you have yours?

MR. WARREN: The blue copy will be filed?

DR. DUPRE: The blue copy, as I understand it, is already marked five. The white copy is marked six.

MR. LASKIN: That's correct.

10 MR. McNAMEE: Well, I can photocopy for anybody who needs five.

MR. LASKIN: I suppose, Mr. Chairman and members of the Commission, it only remains to work out a convenient time when we can call Dr. Finkelstein as a witness, and that may be something that's best done outside the hearing room, after consultation with all the parties and after consultation with the time schedules of the Commission, so that I propose simply to leave it and I will undertake the job of consulting everybody, and with Miss Kahn's help we will arrange a date when Dr. Finkelstein can be called.

20 DR. DUPRE: So it will be my understanding, counsel, that this will be worked out by the parties, and this, of course, includes Mr. McNamee, who represents the Government of Ontario, the party with standing?

MR. LASKIN: Yes, Mr. Chairman.

DR. DUPRE: Thank you.

25 MR. LASKIN: Now, before we get underway, I understand Miss Linda Jolley has one further matter which she has requested to raise with the Commission.

DR. DUPRE: Miss Jolley?

30 MISS JOLLEY: Yes. I wonder...it's not exactly an objection, but we have some concern about the issue of introducing new evidence which we didn't have before the hearings.





MISS JOLLEY: (cont'd.) There was an understanding among the parties with standing that we would receive the papers, rather than written testimony, and Dr. Enterline did present two new papers, one of which the Asbestos Information Association did have and were nice enough to give to each of us, but I think it puts some of us at a disadvantage in questioning last time, and I wondered if we could encourage the Commission to try and ask authors to please submit all the information that they are going to introduce.

We understand that testimony will bring out other information, but entire papers introduced, it's rather difficult to question when you haven't had them in front of you.

DR. DUPRE: Counsel?

MR. LASKIN: Well, I think I should respond to that, Mr. Chairman. I have tried to encourage all of our witnesses to give to me in advance any particular materials they are relying upon, and indeed all of them have basically complied with that.

The two papers my friend refers to are very recent papers, neither of which were published, and I happen to have been in exactly the same position as Miss Jolley. I only saw them when I met Dr. Enterline on the morning of his testimony.

So it was unfortunate, but perhaps my friend, Mr. Warren, is a little more industrious than the rest of us. He seemed to have at least got a copy of one of them beforehand.

We will do the best we can.

MR. WARREN: Mr. Chairman, I might just like to add my word of support to Miss Jolley on this. I understand the problems which Mr. Laskin faces in this regard, but I do think to the maximum extent possible it is extraordinarily helpful for each of us to have the papers beforehand. I think it makes for a better record, it makes it more understandable both for the people sitting in this room, and subsequently for anyone who reads the transcript.



MR. WARREN: (cont'd.) If we have the papers beforehand, I think we can ask more pointed questions and really be more helpful, so I share the concern and recognize at the same time there may be instances where Mr. Laskin has no choice but to go forward the way he did last week.

But I think to the maximum extent possible we ought to try to have the papers beforehand.

DR. DUPRE: Miss Jolley and Mr. Warren, I think it would be fair to say that the Commission certainly shares your views that that convenience is well served by having the material in advance.

On the other hand, perhaps we might recognize that there is one thing worse than not getting the material in advance, and that is not getting helpful material at all. To the extent that our witnesses from time to time will be generous and come, so to speak, bearing gifts, I don't think that we would particularly want to discourage them to do that.

On the other hand, however, I certainly would agree that we should encourage them to bring their gifts, forward their gifts early, to the extent possible. But I would not want to inhibit...and I believe you wouldn't either...witnesses from introducing material that is going to be useful to us.

Is that fair enough?

MR. WARREN: I think I agree with the sentiments that you have expressed. It is occasionally true that when a new study such as exhibits five and six come in, as an example, at the last minute, it's almost impossible for us to ask good and pointed questions, and when we are unable to do so I hope you recognize the limitations that we face when documents come in at the last minute.

I don't mean to point out five and six as egregious examples. I am happy to have them and welcome the opportunity to cross-examine Dr. Finkelstein later this month,





MR. WARREN: (cont'd.) or next month, whenever we can work out a mutually convenient date.

5 DR. DUPRE: Of course exhibits five and six are exhibits which we now have and which you will be able to examine at your leisure.

10 One of the helpful circumstances, of course, is that the witness involved is within this jurisdiction. As for guest witnesses, well, again I would simply reiterate we should try to have the material in advance, but above all, let's have the material.

Any more procedural matters?

15 MR. LASKIN: I think not, Mr. Chairman. With that, I would like to welcome Dr. Hans Weill as our witness here this morning, and I would like to ask Miss Linda Kahn to swear Dr. Weill in.

20 DR. DUPRE: Before Miss Kahn swears in Dr. Weill, may I, on behalf of the Commission, welcome you most warmly. Your reputation precedes you. You have come all the way from Tulane University. We can't accuse you of having brought this weather with you, the fact of the matter is it got here before you did, but I think we probably have this weather in your honour. I hope you feel right at home.

May I now ask Miss Kahn, please, to swear you in.

DR. WEILL: Thank you very much.

25 DR. HANS WEILL, SWORN

EXAMINATION-IN-CHIEF BY MR. LASKIN

30 MR. LASKIN: Q. Dr. Weill, for the record, you are, I understand, a professor of medicine at Tulane University School of Medicine?

THE WITNESS: A. Yes, I am.

Q. You specialize in pulmonary diseases?





A. Correct.

5 Q. I gather you are on the active staff of the Tulane Medical Center Hospital and are a consultant or attending physician at several other hospitals in the New Orleans area?

A. Yes.

10 Q You have been the recipient of numerous honors and are a member of numerous professional societies, and I gather also have been a consultant to the National Institute of Health, United States Environmental Protection Agency, OSHA and NIOSH, and particularly with respect to NIOSH, I gather, have served on task forces on occupational respiratory diseases and on pathology standards for asbestosis?

A. Correct.

15 Q. You have lectured, of course, and written very widely on matters pertaining to respiratory diseases and the health effects of substances such as asbestos?

A. Yes.

20 MR. LASKIN: I am going to file, Mr. Chairman, if I could, Dr. Weill's curriculum vitae and copies of his articles and the brief, as exhibit number seven in these proceedings.

EXHIBIT # 7: The abovementioned document was then produced and marked.

25 MR. LASKIN: What I propose to do this morning, and what Dr. Weill proposes to do, is to give the Commission and the parties an overview, with the use of slides, of first of all, the health effects of asbestos, and second, more particularly, matters pertaining to his own research. Following that, I propose to take up a number of specific issues with Dr. Weill, and then, of course, the parties will have the opportunity to  
30 question.



5 DR. WEILL: I would like to begin by thanking the Commission for asking me to appear and participate in these important hearings. I would like to say at the outset that any gifts that I bear have in fact been published, and are in your hands, in general, I think, so that hopefully you have had a chance to review these. But I will not expect that anyone has so thoroughly digested them to come close to memorizing, and I would be most pleased to clarify or expand on some of the material which almost certainly on quick reading could not possibly have all been clear.

10 The method in which I would like to begin hasn't become absolutely apparent until this morning in discussing with Mr. Laskin how best to approach this. What I have chose to do, with your agreement Mr. Chairman, is just to very briefly remind the audience about the health effects of asbestos exposure in the workplace, and then to go to some data, results of the studies which we have performed in the last decade or so, dealing almost exclusively with asbestos cement products manufacturing workers, but more recently also studying the films of a cohort of marine engineers who have had their asbestos exposure onboard ship in engine rooms.

15 Now, none of that work is, in fact, published, and I have mentioned it only so that people here will know that we have had this experience of reviewing in our unit six thousand films of people who are end product users in an industry that heretofore has not been recognized as having some asbestos risks. Some of this material has been publicized in nonmedical and scientific journals, mainly through the union under whose auspices we did the study of marine engineers, Beneficial Association, and I probably won't mention that again unless there are some questions.

25 Most of our work has in fact been in the manufacturing industry, as I have mentioned.





DR. WEILL: (cont'd.) Now, for the purposes of doing this most efficiently, is there a way that I can run the slides, or shall I just ask you to...?

5 MR. LASKIN: Well, if we move our chairs around a little bit..

DR. WEILL: Would it be easier if I stand? And the other possibility, is there a pointer?

MISS KAHN: I have a ruler.

10 DR. WEILL: All right. Let's just begin and see how we do without any additional devices. It's always nice to...

(REPORTER'S NOTE: There was an inaudible question directed to Dr. Weill at this point.)

15 DR. WEILL: No, I'm actually quite comfortable standing. I might even lean.

I'm glad to see that you have one of the new volumes of the Leone meeting. I hope that they are used for something other than to prop up the desk.

20 The effects, of course, as you all know, associated with the inhalation of fibrous dust, asbestos fibers dust, fall into two categories. They are fibrogenic... that is they cause scarring, and they are carcinogenic or tumorigenic perhaps is a better term...they cause malignant tumors.

25 (REPORTER'S NOTE: At this time there was general conversation about the operation of the slide projector.)

30 MR. LASKIN: Can I suggest we just take about five minutes and maybe organize this slide projector here?

DR. WEILL: There are not that many slides, so





DR. WEILL:(cont'd.) if you want to we can...

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: Mr. Commissioners, I think we are now ready to proceed.

DR. WEILL: We were talking about the fibrogenic and tumorigenic effects of asbestos exposure. This is a chest x-ray of an asbestos cement manufacturing worker, and it shows the first of these recognized fibrogenic or scarring effects, that is, linear and irregular small opacities within the lower lung zones indicating pulmonary fibrosis, and when pulmonary fibrosis is associated with asbestos exposure, it is properly called asbestosis.

This is a later stage of asbestosis, at which time not only are there densities, densities in addition to the normally appearing white linea blood vessels, but there is contraction or shrinkage of the lung which happens in advanced pulmonary fibrosis, there is obscuring of some of the normal structures, the heart being the big white thing in the middle, is very often not sharply outlined. This has been called in some textbooks the shaggy heart appearance.

Asbestosis is a progressive disease. This is an individual who had a very heavy exposure in the forties in our industry, who developed his evidence of asbestosis, pulmonary fibrosis, in the early 1970's. The film on the left was taken in 1972. He had no further exposure after the forties, and developed lung fibrosis. At that time, it was definitely there. It may not appear very striking to you, but as you can see, seven years later in the absence of further exposure, it has progressed.



DR. WEILL: (cont'd.) So similar to silicosis, asbestosis can progress after exposure has ceased, and this is an example of that happening.

5 Now, for those who like to talk about state of the art, question about when we knew about various asbestos health effects, somebody walked into the Health and Safety Executive in Britain and supplied me with this photo micrograph, histopathology showing increased collagen formation, as well as a lot of asbestos bodies. This was presented by Montague Murray to Her Majesty's government in the year 1899.

10 As you know, several years later this appeared in print. So we have known about asbestosis for some time.

15 This, of course, the lungs were from a worker in an asbestos textile manufacturing industry where most of the original information came from.

20 Now, in addition to lung fibrosis, it is well recognized that asbestos exposure, inhalation of asbestos fibers, can also produce scarring, both localized and diffuse, of the lining of the lung, or the pleural surfaces. What we have here is an example of localized pleural thickening. There are two very nice examples of pleural plaque there. I just don't know whether you think a pointer, perhaps...if you'll let me just...on one or two occasions I think it might be necessary to point these out. They occur very often along the lateral chest wall here. It can also occur, these humps in the diaphragm, they very characteristically can occur on the diaphragmatic surface. They are very often bilateral and they are very often indicators of asbestos exposure.

25 Now these plaques and limited pleural effects are in general not associated with clinical or functional abnormalities. Plaques can calcify and then they become very easily visible, very impressive markers of exposure, and if you look at the right diaphragm...on a film the right is on the





5 DR. WEILL: (cont'd.) left and the left is on the right, some of you probably already know that...but if you look at the right daphragm, which is on your left, you'll see those dense, relatively horizontal lines, which are calcified pleural plaques. Very characteristic of asbestos exposure.

10 Now, every once in awhile the pleural process becomes rather diffuse. That is, it's more extensive. This is such an example. Sometimes these occur after benign pleural effusions, sometimes the appearance is without such a collection of fluid in the chest cage.

15 When the extensive pleural disease such as this occurs, there may very well be some effect on the volume of the lungs, some restriction or reduction of lung volume. In that case, for various reasons, perhaps including compensation, one has to deal a little differently than with the isolated plaques.

20 Now, in addition to the fibrogenic effects...and this man, again from this industry, shows those... he has diffused lung fibrosis. Some of you will also see a nodule...this is an oblique projection, which is a little different...some of you will see a rounded density, white area, in the left lower lung. That is a bronchogenic carcinoma. Lung cancer risk is increased in individuals who have had asbestos exposure in the workplace. This is, as you will see in a few moments, dose related....has been in every study where it has been possible to estimate dose, and it is a greater risk in those who also  
25 smoke cigarettes. So that the number of excess lung cancer cases in this occupational setting will be substantially higher than expected, based on both exposure and smoking.

30 This is a calcified pleural...there is no rule that people who have been exposed to asbestos in the workplace can't have more than one effect...this is again someone from the industry that we have studied, who has a calcified pleural



5 DR. WEILL: (cont'd.) plaque. That's that white, sort of dense stuff. This is a plaque seen on-face, rather than in profile, although you can see a small plaque in the diaphragm as well. The major one I am talking about is in the mid-chest wall.

Two years later the plaque was still there, and there was a rounded nodule lesion in the lung which turned out to be another lung cancer.

10 One question which has come up from time to time is, does the presence of that plaque, that other asbestos-related effect, in some way increase or enhance the risk of the ultimate development of this neoplasm or some other neoplasm.

15 I think the evidence that it does is not there. Certainly they both are indicators of exposure. Exposure increases the risk of both, but unless one could do a major epidemiologic study with well defined exposures in two groups, one that developed the pleural changes, say, and one that doesn't, and where exposure is held constant, and show an increased risk in those who have developed this biological effect, increased risk of a more important effect, of course, 20 than malignancy, I'm afraid at the moment we will just have to say that they are both exposure related and therefore have their risk associated with exposure rather than development of one of these pleural plaques, or thickening.

25 Now, the other, of course, pleural manifestation that is recognized and has been for about twenty years in association with asbestos exposure, is a malignant, primary malignancy of the pleural surface called mesothelioma. That can also occur on the perineal surface. Less commonly, but it can occur in the abdominal lining as well. They present in individuals who have had exposure generally in one of two ways, 30 radiographically. One is a large effusion, which is often bloody and which shifts the central structures, the heart and trachea,





DR. WEILL: (cont'd.) over to the other side.

Or like this, where the tumor is just growing around the left lung and encasing it, and in fact ultimately making it airless, invading the mediastinal structures. This again is an x-ray of an individual from an asbestos cement manufacturing industry, with a mesothelioma.

Now, the exposures in the industry that I am referring to have been variable. The object is to make a product. The product is either a shingle or a pipe, asbestos cement pipe, roofing, siding of various sorts, either flat or corrugated, and in the two plants that we have been focussing on, the exposures are highly variable within that plant.

For instance, in the upper lefthand corner is an individual who is opening a sack of Canadian chrysotile asbestos and about to shovel it into this conveyor.

Then, in the upper righthand corner, you can see the fibrous material...the fibers are opened in a willow, and again it is discharged into a wet slurry. That wet slurry contains cement, as well as silica, or sand, and then a few things happen. This material is picked up on a stock and a sheet is made. You can see on the left lower corner a wet sheet which contains, of course, fiber and cement and silica. And also scrap material is continually reground and used again in this mix, another source of exposure, and then it dries, and there is a piece of corrugated siding in the lower righthand corner. It is relatively dry. High energy is applied to it, they use a saw, and again it's a dusty operation.

So one needs to know, and the best way to know is actually to sample for asbestos, which has been done for the last three or so decades in our industry, the industry that we are studying, and we know that there are marked differences in exposure.

Fiber exposure would have been highest in that



DR. WEILL: (cont'd.) man that is dealing with the raw material, essentially a pure asbestos exposure in what he is doing in that picture.

It would have been lowest, perhaps, in these four examples, somebody who is standing next to this machine where a wet sheet is produced. There is very little dust.

And again high where a dry product is sawed, or holes are punched in it at a punchpress, that sort of thing.

The purpose of this is not to make you experts in the production of these materials, which I am not, certainly. But to indicate to you that if one is going to reconstruct exposures, try to develop exposure-response information, you have to know a little about the industry and where the exposures have been high and low, and to quantify those exposures as best as possible.

Now, we did that, and I don't expect anyone to read this slide, but it just is an example of how various job areas have had various exposure levels ascribed to them. This will change, as you can see by the dates. Dust control measures have lowered exposure levels considerably in the last several decades, so that information generated through air sampling has given us then the kind of information that this table shows, that is, say, for somebody in the beater area, which is the man in the upper lefthand corner where he is actually emptying the fiber. Nothing much, unfortunately, has happened to his exposure. It has stayed the same.

It has subsequently, in the seventies, been reduced by certain other dust control measures, but nonetheless, up to age seventy,<sup>(sic)</sup> it was not markedly lower than it had been in the fifties and before.

In other areas, the levels have gone down.

I would also say at this time, in all of our





5 DR. WEILL: (cont'd.) studies we have had to reconstruct individual exposures based on total particulates, similar to what has been necessary in Quebec mining and milling operations, and similar to the exposure reconstructions that you heard about, I assume, on Thursday of last week when Phil Enterline appeared before you.

10 So we have million particles per cubic foot, in terms of average exposure, and then multiply by time in each job for a particulate here or giving cumulative exposure.

15 Now, also you will note here, we have had our population exposed to varying types of asbestos fiber, and in fact in this industry chrysotile, crocidolite and amosite have all been used. I'll get back to that to provide some data, some information about possible different health effects associated with these fiber types, but we have been able to estimate whether or not exposures have been only to chrysotile, or to a mixture of chrysotile and amphiboles.

20 Then ultimately this is just a simple example. Many of our workers have had much more complex work histories than this, but here's somebody who has spent a year or so, a year and a half, in the beater area and then went to another area, and so forth, and for each of those we were able to generate information concerning his exposure in that location at that time, and we've come up with various dust exposure indices.

25 Now, the first phase of our study began around 1970. Data collection began around 1970, and was a cross-sectional study. That is, everyone in the working in two plants at a given time was included in a morbidity cross-sectional study. These were active employees who had had on an average seventeen years of exposure by that time.

30 They each were studied in a laboratory, they had chest x-rays performed, they had lung function measurements done, questionnaires administered, a job history was generated, and they had their exposure profile reconstructed, as I have



DR. WEILL: (cont'd.) already indicated.

Now, let's look at the lung function, first of all.

In all of these, the next several slides, we have divided...this is a group, by the way, of between nine hundred and a thousand workers. This is all in the materials that you have.

We divided this group up into five exposure categories from left to right, and on this first slide we are looking at lung function, lung volumes, in relation to their exposure, and provided whether or not they had any x-ray changes of pneumoconiosis as well.

If you look at the first panel up and down, these are all...let's just not worry about the compartments necessarily...these are total lung capacity, it's the entire volume, vital capacity and functional residual capacity in all compartments.

In each instance..and let me make one little comment about how the data are presented...percent standardized could be considered to be percent predicted. We have looked at this in terms of predicted formulaes...all the predicted formulaes, actually, and that appears in, for instance, some correspondence that came after this paper, and they all show the same thing.

We chose percent standardized...that is, looking at the experience of this population in relation to that same cohort, the internal group of people who were minimally exposed, who had no x-ray changes, no respiratory symptoms, so that the data are presented in relation to that group.

This is a recognized and suitable...there is a little bit of tradeoff whenever you present any cross-sectional data lung function studies in relation to what the expected is, what should they be. You can take a control population from Tucson, Arizona, or from the Veterans Administration Hospitals around the country, or somewhere, and you have the problem then,





5 DR. WEILL: (cont'd.) well, is your group in New Orleans or Toronto comparable to the expected. You eliminate that largely by using an internal standard. You don't eliminate the possibility that even your minimally exposed population may have had some effect, and we think they do not, but that's a matter perhaps that you might...

10 Anyway, in each of these lung volumes I think you will see a very substantial dose-relatedness as the levels of exposure, cumulative exposure, increase, the reduction in lung volume increases as well.

I'll deal with smoking in a moment.

You can also see that if you divide people up by whether they had x-ray changes or not, certainly those with x-ray changes had lower lung volumes than those who did not.

15 Those little circles or stars above the bars indicate statistical significance and three of those stars, or whatever, indicate a P value of less than point double 0 one.

20 Now, these are the expiratory flow rates, with forced expiratory volume in one second, again shows dose relatedness. Higher levels of exposure - lower, significantly lower FEV 1.

25 Midflow is the one on the bottom, which also shows a significant dose relationship, but the ratio FEV 1 over VC does not, and I think most of you will recognize why that is, because both the numerator and the denominator are decreasing with increasing exposure, and therefore the ratio doesn't show this relationship.

30 Now, finally, if there are some addicts of the diffusing capacity as a measurement of early effects of asbestos exposure...and there are such, centered mainly around Boston - my good friend Ed Gainesworth thinks it's a very good measurement for early effects, except he doesn't have



5 DR. WEILL: (cont'd.) data of these types to really be sure of how it relates to exposure. But nonetheless, we did not find that the diffusing capacity...and see there is no dose relationship...the diffusing capacity is very good as an early indicator...this is a healthy working population...as an early indicator of asbestos effects.

10 We did find that the diffusing capacity separated out those who had x-ray changes from those who didn't, but by that time we really didn't need that.

15 I might say to you that the diffusing capacity has its reputation as a good test of alveolocapillary membrane surface, or what have you, because of its abnormality in individuals who have well established disease. In the days when it was important and popular to, say, take twenty people with asbestosis, recognized asbestosis, measure their lung function, certainly diffusing pressure is going to be low. So will a lot of other things. on volumes and so forth, but as an early, sensitive indicator, which is really what we are most interested in, it didn't work out too well.

20 There are some exercise studies on the bottom which again did show some dose relatedness, but not very much better than the simple spirometric measurements.

25 As a matter of fact, that sort of is the message here, that properly done, standardized and properly performed, spirometric measurements in our hands were as good as anything else in detecting an early effect.

DR. UFFEN: Can I interrupt?

DR. WEILL: Yes, surely.

DR. UFFEN: Pardon a laymen's...

DR. WEILL: Of course.

DR. UFFEN: What is spirometric?

30 DR. WEILL: Measurements of timed volumes and flows. It is the simplest type of lung function...if you go to





5 DR. WEILL: (cont'd.) for a pre-op evaluation, lung function, you will get spirometry...which you will, hopefully, but somebody will get spirometry...blow into a machine, it may have a bellows or it may have another device now, an electronic thing, and all it does is measure the volume that you exhale, and it gives you the time it takes to do that in various breathing maneuvers. It's the simplest measurement, and in my view in many of these occupational lung diseases, nothing else has been shown to be very much better.

10 I'll be interested to see if David Muir has some other comments on that anytime.

15 Okay, now, you do have to, obviously, when you try to relate lung function with exposure, you have to account for confounding factors, and all of you heard...perhaps ad nauseum...that an important confounding factor is smoking. Smoking clearly, in some individuals, affects lung function.

20 I think in our studies you can see how it does that, and this again is from the cross-sectional data, and it's the decline in the mid-flow over the two decades from age thirty-five to fifty-five. You can see that the nonsmokers in the line on top there, decline almost not at all...very, very little.

The exsmokers have an intermediate position, and the current smokers have the steepest decline. This has been found by others as well.

25 Now, how might that influence results? Well, if you don't account for it, if you have maldistribution of smokers in various exposure groups...let's just say for example you have all your smokers in the low exposure group, or a higher proportion than in the high exposure group, that lowers the lung function of the low exposure group and you may say there's no exposure effect.

30 Or you could have it the other way. Let's



DR. WEILL: (cont'd.) say you have most of your smokers in a high-exposure group and you are giving exposure then the credit for some of this lung function.

5 So now there are many statistical techniques, very good ones, various ones, that can account for such confounding practices as smoking.

10 Now, smoking isn't the only thing....atopy, for instance, the predisposition for allergic respiratory disease is another. But certainly one needs to take these things into account.

15 Now, the next thing we did in order to find out what the determinance of progression of lung function and x-ray changes is, we took part of the cross-sectional population, we defined them by age. Everybody at the time we first began this study between the ages of forty-five and fifty-nine, were asked to participate in a three-yearly examination which, up to this point here, was six years, and now is nine to ten years, and those data are in the mill. These have already been presented and published, and again you have had some of this information.

20 We selected this slightly older group for two reasons. One is, we wanted to maximize the possibility of finding an effect so that we could see what the determinants of that effect are, and secondly, we wanted to have as stable a group as possible, figuring that if somebody had been in...was age forty-five and had been in the industry for some years, that they would likely...and in fact in this case the average was about twenty-some-odd years, they are likely to stay there for a little while longer. They are not likely to move on.

25 So that was our population.

30 As you can see, our follow-up was pretty good. Forty were not available for a second film reading, and of those forty, twelve in fact were dead - one of lung cancer and the



DR. WEILL: (cont'd.) others of other diseases, mainly cardiovascular problems.

5 There is not much difference in those who were lost to followup from most things. In fact, the few...there was more chronic bronchitis among those who were lost to followup, they had a higher prevalence of smoking. Age was not very different, time since first exposure, and length of exposure not very different. Average in cumulative exposure very comparable in this.

10 This whole exercise is to hopefully add some validity to the results of those that we were able to followup. We don't think that it was very biased.

15 It is true that those who were lost to followup had a slightly greater prevalence of x-ray abnormalities at the first time.

20 So if anything, we may be slightly underestimating the longitudinal effect because slightly...well, sicker if you like to use that term...people were not available either because they died or because they were not available for some other reason.

25 Now, what did we find? First of all, x-ray readers independently judged whether or not, on serial films with order known, whether there was progression of the x-ray finding. There was a five point scale, actually, even along to the reading of regression. There were three readers, including John Gillson from South Wales.

30 The majority agreement was very good, but in order to call somebody a progressive we required that only one out of the readers in fact felt there was definite or probable progression.

Now, this really tells pretty much the whole story on our progression analysis to date. Small irregular opacities, if you allow me this slight liberty...it's not much of one...we could equate with asbestosis. This is the





DR. WEILL: (cont'd.) characteristic radiographic finding of pulmonary fibrosis, or asbestosis.

The other two things that made it quite self-explanatory, pleural thickening and pleural calcification, the latter being a later stage, as I have already shown you.

Well, first of all we found that progression of age, progression of these findings is not related to age. This was a possible influencing variable. So age did not significantly influence...and this was done by logistic regression analysis, if any of you are statistically inclined. Age did not influence progression.

Smoking did not influence progression. Now, smoking and shadows on the films - that's an area that has received some attention. There are people whose studies seem to show that there is an effect of smoking on the risk of having small irregular opacities. Our studies in fact did not show that. Ours did not, and some have. I think it's an open question.

But as far as progression of these densities, either pleural or lung densities, smoking did not influence them.

Now, length of exposure was the important determinant of progression of pleural thickening or pleural calcification. Average exposure and cumulative exposure, which is a better indicator of dose, did not significantly influence progression of pleural thickening.

Average exposure and cumulative exposure, however, was highly significantly correlated, again  $P$  is less than double 0 one, highly significantly correlated with progression of small irregular opacities...or if you will, asbestosis.

Now, what does that mean? It means that in this study today, six year analysis, bronchotumor analysis followup, it means that progression of asbestosis seems to be



DR. WEILL: (cont'd.) dose related. Progression of the pleural abnormalities seems to be time related.

5 I might say that more recently these findings have been replicated by your colleagues in the province to the east, in Quebec, at McGill, where this relationship of length of exposure and pleural changes and dose, either cumulative or average exposure, and small irregular opacities progression held as well. So I think it's real.

10 It doesn't necessarily mean, of course, that you don't have to have some critical dose to reach the threshold, perhaps, or the level at which pleural effects occur, but once having reached whatever that dose is, progression of pleural changes, and indeed almost certainly the appearance of pleural changes, will depend on time rather than dose.

15 Now, that has some implications, and I would prefer to wait with those until we perhaps get into a discussion period. We could discuss it now.

It has implications about removing people from exposure, and it probably also has implications in terms of prognosis. So we'll perhaps discuss that later.

20 As statisticians love to do, they can take these data and develop probabilities. Based on what I've just told you, Dr. Deem in our shop has shown that as...let's just take small irregular opacity progression, fitted probabilities of progression over the six year period. So that if you are in the highest exposure group, in this case the average exposure being thirty millions particles per cubic feet, your probability of progression over that period of time is almost thirty percent, and of course it goes down.

25 In the pleural thing, we've always said are time related, and again you can see after thirty years in this industry you are likely to have progression of pleural changes. Again about a third or so of the individuals will

30





DR. WEILL: (cont'd.) exhibit that. About half that probability for pleural calcification.

5 Now, lung function as well in time is related to cumulative exposure. The most stable of the indices here are forced volume capacity and the FEV 1, but in this case decline, average annual decline in lung function is also importantly influenced by smoking. Remember I mentioned that in our population smoking didn't influence x-ray progression, but it clearly has an effect on lung function decline, which is 10 not surprising, so that in this instance both smoking and exposure to asbestos dust in this industry will have an effect on those two parameters, not reaching significance in other of the lung function measurements, close to significance for total lung capacity correlation with cumulative exposure.

15 Finally, in terms of the longitudinal study, it will come as no surprise again that there is a relationship between those who progressed on x-ray and those who decline most on lung function. That's all this shows, that the progresses tended to have greater annual decline in function than nonprogressives.

20 I might caution you though, this is what analysis of this population showed. In an individual case it is at best hazardous to predict what is happening to lung function by looking at the chest x-ray. On individual cases, it would be best not to do that.

25 Now, to the third phase of our interest, and that is the mortality study. The mortality study is designed to look at all past employees of these two asbestos cement manufacturing plants, and for reasons that I can assure you were not sexist, we limited this analysis to males, because there were too few females to make the analysis worthwhile.

30 We wanted to look at the effect of minimal exposure in terms of length, many years ago, so we had a



5 DR. WEILL: (cont'd.) minimum of one month employment for entry into the study, and as you know cancer risk most often doesn't appear until twenty years, so we also limited the final cohort to those who had had at least twenty years of followup.

10 These criteria gave a cohort of about five and a half thousand individuals, sixty percent of whom were employed for less than a year, and eighty percent of whom were initially employed in the 1940's.

15 We, for reasons that are explained in our paper, had to rely on Social Security reporting for these...for reporting of deaths, and they reported six hundred and one deaths in this total cohort.

20 Now, Social Security knows the following things about anybody that you ask them to provide you with information about: they know who has submitted a death claim, they know who they are having a financial transaction with...that is, they are either paying a disability payment or paying a pension, old age pension...or they are getting money from them because they are still employed and the employee is contributing to the system...a financial transaction was done.

25 Then they have a third group that they don't know. They simply do not have either an ongoing financial transaction and they have not had a death claim.

30 Now, one of the problems here was...and I again won't go into this in great detail, but we can discuss it if some of you are interested...one of the problems was that this study came at a bad time in terms of our local economic situation. We found out that most of the people, about twenty-five percent actually, who were not having financial transactions were in the age group forty-five to sixty-five, we know that there were, including in this industry and in other industries, tremendous layoffs at that time. These were



DR. WEILL: (cont'd.) people of an age not likely to find another job, and in fact that was the case, so that a number of these people were lost to view.

5 Now that's not good in a mortality study, and we were concerned about it and did a lot of thinking about how we could best estimate whether or not these people were in fact alive. We did make that estimate and they were in this analysis, assumed to be alive, and I'll show you the results in a moment.

10 We were able to trace ninety-one percent of the death certificates and in exposure categories, those where a death certificate was not available, cause-specific deaths were allocated in the same proportion as those where deaths were known.

15 Again, we took individual exposure information based on work records and measurements, as I've already said. We got the cumulative exposure and we got various fiber type groupings.

20 The followup within these five exposure groups then was very similar, so results could not be explained. Differences in exposure groupings as far as the mortality experience could not be explained by significant differences in followup. There was a slightly longer followup in the highest exposure group, but as you can see it averaged about twenty-seven to twenty-eight years all across.

25 Now, we did not find a dose-related excess of specific mortality in this population, except for respiratory malignancy. Specifically, along with other studies such as the Peto, Roachdale study and the Dement textile study recently reported but not yet published, we were not able to find an excess in gastrointestinal malignancy. That's a point we may want to go back to.

30 Now, our SMR's, our standardized mortality ratios, tended to be low, and they were a little lower in the low-exposure





5 DR. WEILL: (cont'd.) groups...this is overall mortality, which are solid lines around the dots. Now, overall mortality was lower than a hundred standardized mortality ratio or relative risk of one...you can use either that you are more comfortable with...and tended to go up with exposure.

10 Well, along with that going up, we also had a better trace, a much higher trace, among the high-exposure groups than the low-exposure groups, which might lead to the question... and this deals with place again, might lead to the question, are we underestimating mortality in the low-exposure groups because our trace rate was less satisfactory than it was in the higher?

15 Well, in fact, that is possible, and in fact I would even say it's probable. What one would have to do then is make the following assumption: Since the trace rate was very good up here and the overall mortality is just about where it ought to be in one, why not say that we have underestimated mortality and bring these close, the overall mortality, close to where it should be?

20 The second assumption...and you would then have to raise the respiratory malignancy mortality by about the same proportion, something like a third more, which we can certainly do.

25 The other assumption is that there should be no relationship between trace and cause-specific mortality. I mean there isn't any reason, to a degree or otherwise, to suspect that people who have been lost to followup are any more likely to die, say of asbestos-related disease, than they are anything else. There is just no reason that I know of, we thought very hard and we had a lot of help in thinking about this, people who believed in the credibility of this and others who perhaps had some doubt, but no one has come up with any reason why there ought to be a relationship between trace and  
30 cause of mortality.



5 DR. WEILL: (cont'd.) Well, if we bring up the respiratory malignancy, relative risk for SMR's also up about a third, you still will see that in the low-exposure...and these are a very low-exposure category...the SMR's or the relative risks for lung cancer are not elevated in those low-exposure groups.

They most certainly are elevated in the moderate and high-exposure groups.

10 Now, we are in the second year of an update of this mortality study, and we have several important advantages this time. One is that we have more deaths. There is at least fifty percent more deaths in this five year period since the cutoff here, and second, the Social Security regulations on confidentiality have changed so that now, instead of their not being able to tell us who they are not having a financial transaction with, they now can tell us that so we can actually  
15 do a trace of what now amounts to...and this is ongoing...those thirteen hundred people rather than the whole population of five or so thousand that were not known to be dead last time, which would be an impossible task.

20 We are now able to focus on another group that we can find out about via status.

25 So, unfortunately this is going to take another year or two before any of the results are in, but our trace rate would be far better and we may very well also have a tightening up of the ninety-five percent confidence limits in regard to the dose-related excesses.

30 Well, so much for that. Now, there is no question that above a hundred million particles per cubic foot years, which is a lower level than, say, the Quebec level, beyond that there is a two to three times risk of respiratory malignancy. There were a couple of mesotheliomas in this population, probably underdiagnosed in the past years.





DR. UFFEN: Excuse me.

DR. WEILL: Yes, sir?

5 DR. UFFEN: That reference to Quebec, you are talking about two different measurements. One that we've heard about before is two fibers per c.c. If I understood you correctly then, that below two fibers...or above two fibers per c.c. the rate would be quite significant?

DR. WEILL: Okay...

10 DR. UFFEN: It's that translation from particles per cubic foot year, or whatever it is, to...

DR. WEILL: Right. Let me deal with this at this point. I have before the discussion period one or two transparencies that deal with it in greater...

15 Both our studies and the studies of MacDonald in Quebec, had as their basis for exposure this measurement - total particulates over the years past, which of course was when these cohorts were exposed. They have tried, and we have now tried as best we can, because our modern standards are in terms of fibers rather than total particulates, have tried to convert what was the exposure if we were to use modern methods.

20 Two comments about that: We both have tried and are doing it to a certain extent. We are doing it with considerable trepidation, it's a risky business and I want to show you, if you have time, during the discussion, just what our conversion information tell us. But for the moment, I'm afraid, what I've told you so far deals only with total particulates.

25 Now, in the MacDonald...the latest paper...they have made this conversion, and we can make it too in a few moments, if you like.

30 All right. Now, one of the things that you may hear from other witnesses, and perhaps have already heard today,



5 DR. WEILL: (cont'd.) is why go through all this Mickey Mouse stuff of reconstructing exposures? Why not just look at years of exposure? I mean that's a surrogate for exposure or cumulative exposure. It certainly is a component. Cumulative exposure is a product of time and average concentration.

10 We and some others have always felt that simply time of exposure is not enough, because people are exposed at different levels. Some jobs, as I've showed you in our industry, are relatively low-level exposure, and some are very high level exposure.

15 What we did in the mortality study then, was to see whether or not both duration of exposure and average concentration play a part, and I think you can see quite clearly that both do...that relative risk goes up as you go to increase in durations of exposure, and goes up as you go down to increase in average dust concentration. Infact, it appears that...well, both are important. I would say that they both play an important role.

20 Again, this has been suggested I think by Phil Enterline, and there is no data that I know of that refutes this. So years of exposure, that's one element of dose, while average concentrations, which may have changed over those years but which can be summed for various jobs at different times, is another.

25 Now, this puts together...before we get to a final consideration of fiber type...this puts together some information about...from our population...about both asbestosis or indicated with fibrosis, lung function and x-ray indicated with fibrosis, as well as mortality. To try to get some idea... one of the questions is, what dose increases the risk for asbestosis vis-a-vis what dose increases the risk for lung cancer? You can get all kinds of opinions on that. Most of

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DR. WEILL: (cont'd.) it without data.

5 In the top two...I've showed you all of this data already, I've shown you all of it. The top two has to do with the x-ray abnormalities, particularly in irregular opacities and probably most specific for asbestosis.

10 Let me just say the reason we also have rounded opacities, for the physicians in this group, is that there was a substantial silica exposure in the industry and that almost certainly is playing an important role, and we have some evidence of that, in fact, that it is playing an important role.

15 Anyway, dose again, five exposure categories from left to right, these are lung function changes after smoking had been taken into account, and this is respiratory cancer risk, again on the same exposure axis.

20 What can you see on that? Well, what I see on that, and you can perhaps see, is that the elbow of the dose-response curve appears at about the same time, doesn't it, or at the same dose, I should say, and that is about a hundred million particles per cubic feet. Dose responsiveness or dose relatedness starts to appear at that cumulative level of exposure.

25 Now, let me remind you of something, and that is that the morbidity population was studied after seventeen years of exposure because they were an actively employed group. Had they been studied ten years later, almost certainly more of them would have had x-ray and lung function changes of asbestosis, which would have moved the curve over in that direction and they probably would have shown the elbow in this curve at even low levels of exposure. Remember that the followup of the mortality study was twenty-seven years, and not a decade longer. So if anything my thesis is, in this industry that it appears that the expiry evidence and lung function evidence of asbestosis is likely to appear as doses

30





DR. WEILL: (cont'd.) at least as low, and perhaps lower than, the risk of lung cancer in this population.

Now, this doesn't perhaps finally prove it, because really we are not looking at the lungs, asbestosis, end point for lung cancer is almost certainly better than looking at surrogates, knowing what somebody died of, x-ray and lung function, but I think there is awfully good evidence that in fact if we ever got down to a level of exposure that after a suitable period of followup, of working lifetime at least, there was no evidence of asbestosis, no evidence radiographic or any other way of asbestosis, it seems to me that at least in this manufacturing industry one would probably not find excess, detectable excess risk of lung cancer.

Now, we'll get back to the shape of the dose-response curve again, probably, in the discussion, and what we know about that, but let me just say detectable excess risk of lung cancer.

Corbett McDonald's study...again, I always think it's good to contrast or compare certain studies...in fact has low exposure groups where he even has mortality from asbestosis, and yet no mortality from lung cancer...excess mortality from lung cancer...which would, of course, support this thesis.

So again I bring this out and perhaps emphasize it a bit, because I have heard and perhaps you have heard that standards in the past have been set to prevent asbestosis, with the implication that even if you prevent asbestosis you almost certainly won't prevent the excess risk for lung cancer. I am not sure that's correct. As a matter of fact I suggest that it probably is not.

A very controversial subject that I'm sure some others will have questions of in discussion.

Finally, we get to...do I have another just a few



DR. WEILL: (cont'd.) minutes, do you think?

MR. LASKIN: Sure.

DR. WEILL: I just thought it a little late.

Let me just deal with the question of fiber type.

Again this is a controversial subject. There are people who have ideas on all sides of this issue, the question being is one fiber type more hazardous than another, and if so, for which biologic effects?

In the middle is chrysotile, on the left is amosite, on the right is crocidolite. You all know that. I think you even have some of this stuff around here. You know that this is serpentine rock from which chrysotile comes, and this is an advertisement for blue asbestos that appeared, I think, about 1890, which is when it began to be used. It's really very good stuff commercially, apparently. I'm told it has some very important properties, but I think it caused perhaps some more problems than some of the other types, particularly that you mine in this country.

Now, I'm going to go over this aspect a bit hurriedly. This was stuff that was presented in an international meeting, inhaled particles, several years ago in Edinburgh, but let me just say that in our population we had an opportunity to segregate or categorize a group that was exposed to both chrysotile and substantial amounts of crocidolite, blue asbestos, in the manufacture of asbestos cement pipe.

We also had a population that had never been in the pipe plant and had never had blue asbestos exposure, because we took out the maintenance people in these kinds of analyses, and we compared various outcomes in these populations.

The first thing we did was look at the indicated or asbestosis. This is prevalence of small irregular opacity, and without boring you with all of the details here, our conclusion was that the dose relationship was best for the





DR. WEILL: (cont'd.) crocidolite-exposed individuals .....to that portion of the fibrous exposure which was indeed crocidolite.

5 Again, it was...it is not conclusive, but it is cited in the Simpson report and I think it's about the only evidence that blue asbestos may have a greater fibrogenic effect.

10 Now, that could be due to a number of reasons. It could be due to physical dimension of a fiber, but it could simply be due to the dustiness of the fiber. There are some people who suggest that the addition of blue asbestos just makes it dustier, and that may be the reason. But whatever the reason, we feel that asbestosis, including the x-ray, and this shows the lung function differences after total fiber exposures, which were kept constant, the attached bars  
15 or the percent standardized for these various lung function parameters in the pipe workers and those who never worked there, and again there were significant differences, and let's not dwell on them.

20 So we thought there were some differences there. Now we also looked in our mortality study at that part of the cohort of past workers in this industry that had either intermittent or steady employment in the pipe plant, intermittent would generally be maintenance people, steady would be production people, and those who had never been exposed. Again you will see some of the highest relative risks in those  
25 who had, these numbers now, of course, become low and we have tried to keep total fiber exposure constant in the three categories above, and we tried to look at this in relation to whether or not blue asbestos-exposed people had a greater risk, and we thought that they did, and this risk was increased particularly in those intermittently exposed.  
30

Now, this may be confounded by the fact of



DR. WEILL: (cont'd.) intermittent exposure. There are those people who feel that intermittent high-level exposure may in fact be more hazardous than moderate or low-level continuous exposure for such reasons as overwhelming the defence mechanism and so forth. So there is that question in interpretation.

But again, we felt that this was suggestive, at least, that the people who were exposed to crocidolite had a greater risk.

THE FOREGOING WAS PREPARED  
FROM THE TAPED RECORDINGS  
OF THE INQUIRY PROCEEDINGS

Edwina Macht  
EDWINA MACHT

...to page 39



DR. WEILL: (cont'd.) Now, finally, credibility; about how exposures are reconstructed.

5 One day, Dr. Margaret Becklake (who I think you will be hearing from later also) and I were at a meeting and we each presented things. She thought it would be fun to show this, which is a dose response curve for small, irregular opacities in our various populations. Hers was the Quebec mining and milling population and ours, the asbestos manufacturing group.

10 And particularly in one of the mines, just look at the amazing similarity of the dose response curves for those two populations; really quite remarkable, I thought, particularly in view of the fact that we have different types of exposures.

15 And, finally, just before our mortality paper was published, this famous plant in Rochdale -- and I guess you'll be hearing about that from Geoffrey Berry later this week -- a mortality study by Julian Peto was published in The Lancet, where we compared -- and this is now asbestos textile manufacturing in the Midlands of Britain versus asbestos cement manufacturing in New Orleans, and if you look at the two populations, minimum of ten years' employment, minimum of twenty years' follow-up, the relative risk is almost remarkable.

20 I think if I were looking at this, I'd look at it with some suspicion, because it's so close. So it does show, I think, with all the imperfections of epidemiologic studies in trying to assess risk in different types of asbestos exposure, the comparability in the findings of the studies are greater than the disparity. And, again, I'll have some more comments about that.

25 Sorry; I've probably gone just a little over an hour, but ...

30 MR. LASKIN: Thank you very much, Dr. Weill.





MR. LASKIN: (cont'd.) It might be in order, Mr. Chairman, if we all took about five minutes.

INQUIRY RECESSED

.....

INQUIRY RESUMED

DR. DUPRE: Counsel, are you ready?

MR. LASKIN: I think I'm ready, Mr. Chairman; thank you very much.

Dr. Weill, can we start with your cross-sectional morbidity study, and can I just ask you a few questions about methodology.

There's -- in one of your articles, your article at tab 7 in the exhibits. Let's just refer to them by the tab numbers, and I won't have to make a long recitation.

If I can take you over to page 352 of that article.

THE WITNESS: This is tab what?

MR. LASKIN: Seven. The top of the second column, in the first full sentence, you say the risk of non-malignant health effects of asbestos is not assessed by studies of cause specific mortality. A considerable portion of the spectrum of pulmonary and pleural fibrotic disease is not directly associated with a fatal outcome, and is therefore not listed as the primary cause of death.

Q. And are you suggesting that it's not infrequent that a person may have asbestosis but ultimately die of something else, and that something else will then be listed on the death certificate?

A. I'm suggesting not only that it isn't infrequent, but that would be -- particularly in the times we're



A. (cont'd.) living in now -- would be likely.  
The probability of that is substantial.

Q. Can you expand ---

A. Sure.

Q. --- can you give some examples of what kinds  
of diseases are we talking about that might -- what kind of  
deaths?

A. The people that -- workers who have been  
exposed to levels prevalent in at least parts of the industry,  
major part of the industry -- manufacturing, for instance -- in  
the last several decades, two or three decades, have been  
exposed to levels below what they were at one time. I mean,  
there has been an impact on that.

Part of that impact is that the changes of asbes-  
tosis that we see radiographically and that we impute to some  
of the lung functional abnormalities and, for that matter, even  
that are seen pathologically on biopsies, or whatever, or  
changes at the lower end of the spectrum in severity.

As a matter of fact, I suppose there's been some  
introduction of the concept of the international classification.  
I would say that the vast -- I mean, the great proportion of  
workers that I evaluate, either in the course of research or in  
the course of doing individual evaluations -- the numbers are  
fairly high -- are people who have, when I make a diagnosis of  
asbestosis, grade one disease; category one disease.

Now, that means that the likelihood, even though,  
as I've shown, the disease will progress in relation to dose --  
the likelihood of that disease having a fatal outcome from res-  
piratory failure, which is of course what this would mean, is  
rather low.

They will -- they may be impaired by their asbes-  
tosis, they may be limited in terms of functional capacity,





5 A. (cont'd.) exercise tolerance, and so forth, but the prospect of that being a primary cause of death is going to be increasingly low; and, therefore, if you want to study the impact of asbestosis in relation to more recent exposures, this statement leads to the conclusion that you should study it by some way other than waiting for it to appear on the death certificate as a primary cause; X-ray, lung function, and so forth.

10 Q. Are you also suggesting, though, that pre-existing asbestosis makes you more susceptible to whatever fatal outcome there may be?

15 A. Well, I'm not excluding that possibility. If, in fact, there is limited gas exchange, limited oxygenation, it is certainly not inconceivable that somebody who has cardiovascular disease or some other problem in the cardio-respiratory system will have that problem enhanced, or made worse, by the pre-existing chronic fibrotic disease of the lungs. I certainly would say that that is a plausible scenario.

20 For instance, if somebody with asbestosis, maybe not severe enough to end terminally by itself, develops a severe infection (pneumonia), that person may -- will have less reserve, lung reserve, and may have a more serious, potentially fatal episode, and it may not show up as asbestosis but may show up as pneumonia or heart failure, or something else. Now, of course, it's a matter of degree.

25 At the lower end of the spectrum -- and I've already suggested we've seen a lot of that kind of disease now -- at the lower end of the spectrum, very often the functional implications, although discernible in a population study such as ours, from a clinical standpoint are relatively insignificant. So it depends on how severe the disease is; but your point is well taken on ---

30 Q. Are you yourself, as a physician, seeing that



Q. (cont'd.) kind of thing?

5 A. I'm really not seeing very much advanced asbestosis at the moment; I'm seeing mainly disease that has X-ray characteristics. I've seen some malignant disease, of course. But very rarely do we see asbestosis of a degree which either is so functionally severe that respiratory failure and death is a likely outcome, or that it has made a major impact on other cardio-respiratory conditions. But I think it's certainly possible that it might still be happening.

10 Q. If you assume that somebody's got this grade one asbestosis, as you call it, and then dies from something else, what does the physician put down on the death certificate?

15 A. It probably varies, at least as much as we -- the physician is working and, of course, individual variability among physicians, whether or not an autopsy has been performed and a diagnosis is established.

20 Many physicians, treating physicians, are not aware of people's occupations, unfortunately, and the idea of an occupationally related disease doesn't even come up. I'm afraid that, even when that awareness is there, most commonly it would not be mentioned, except by certain sensitized health care delivery people.

Q. Under what circumstances would you yourself mention it, if you had that kind of case?

25 A. Well, I would mention it under two circumstances: one, if an individual had severe lung fibrosis due to asbestos exposure and he died, or she died, of respiratory failure, I would consider it a primary cause of death.

30 If the other scenario were operative, in that another type of cardio-respiratory disorder occurred and it was my judgment that the lung fibrosis played a part (made things worse, if you will, made oxygenation more difficult, sustaining



life more difficult), then I would say it was a contributing cause.

5 Q. Let me turn to something else which you mentioned in your talk. As I understood that little chart that you put up, which related progression to various factors, and I think your chart's reproduced in one of your articles at -- I think, at tab 10, page 540.

10 Correct me if I'm wrong, but, as I understand, one of the things that that chart shows is that there's no statistically significant relationship between asbestosis -- or progression of asbestosis and length of exposure.

A. Length of exposure alone; that's correct.

Q. All right.

15 A. Just to add one sentence, length of exposure, since it's a component of cumulative exposure, does play a role, but simply length of exposure we did not find to significantly relate to risk of progression.

20 Q. Does that mean that if you've got a particular employee in the work force and he's obtained a certain cumulative dose, and he's got some evidence of asbestosis, that if you take him out of the work force completely that there's going to be no progression of that asbestosis, or no statistically significant progression?

25 A. No; because the risk of progression is going to be determined by his past cumulative exposure, and the greater that cumulative exposure up to the time that you've taken him away from exposure, the greater his risk of progression.

30 Q. So that by the effect of taking him out of the work force, then, I take it, is to slow down the progression because it's only then responding to cumulative dose; it's not responding to further exposure?





5 A. You can't eliminate progression; the purpose of -- and why I've recommended that such individuals, with evidence of asbestosis, be taken out of the work force is because, by keeping him in, in an area -- this is assuming that he's continuing to have some exposure, albeit at lower levels -- you ultimately produce incremental exposure, so that even at today's levels of exposure, in twenty years today's levels will have added to that cumulative exposure, and progression risk, at least theoretically, may have been increased by that additional exposure.

Is that clear?

15 Q. In laymen's terms, does it mean -- in my simple terms, does it mean that you will slow down the rate but you will not eliminate the rate by removing a person from the work force who has some evidence of asbestosis?

20 A. I think that's a reasonable way to look at it; I think that's all right. It's not exactly the way we say it, but in fact what you're doing is minimizing the risk, or keep the risk from getting as high as it might otherwise get with incremental cumulative exposure.

25 Q. In any event, that's one of the, I take it, preventive treatments that you as a physician recommend for someone who has evidence of asbestosis; is to remove that person from further exposure?

30 A. I do. I don't do it with the naive impression that I'm having a major impact on his disease, because in fact most of the injury, or injurious exposure, has already taken place; but I do it because I feel that, since progression is dose-related, I don't want him to get any more dose. I mean, that's basically the bottom line.

Q. Is there -- apart from that kind of preventive treatment, I mean, is there any positive treatment that a



Q. (cont'd.) physician can administer to asbestosis; is there any new developments in medicine that allow for better treatment of this particular disease?

A. Only the complications. Certainly there's nothing that I know of that can be done for the scarring of fibrosis.

Q. Your nine hundred-odd employees that you did a cross-sectional study of, are you continuing to follow the entire cohort?

A. At the moment, we're not following the entire cohort; we're just following the sub-population that I mentioned that forms the longitudinal cohort; those people who were, at the time of the initial study, cross-sectional study, between the ages of forty-five and fifty-nine, which represents about a little more than a quarter of the total group.

That's not to say that it may not, at some point, be useful to go back and look at the entire group; but, at the moment, our longitudinal study is limited to the population I mentioned.

Q. I take it, it's fair, in respect of that particular study of some nine hundred, that you wouldn't -- the extent that there were a number of people in that group that were recently employed and you wouldn't necessarily be seeing the latency effect of whatever evidence of asbestosis there was?

A. That's absolutely right.

I have a few additional comments on latency at some point, if you want. There is a latency period for all of the asbestos-related effects -- certainly that includes asbestosis -- and those who would have been most recently employed and would have accumulated the least lowest exposure would not be expected to have it.

And, indeed, part of the dose response curve in





A. (cont'd.) that cross-sectional is related to time as well as dose; absolutely.

5 Q. Okay. One or two other methodological questions; dust exposure histories, which I think you referred yourself in one or two of your -- places in your articles, how difficult a task it is to reconstruct dust exposure histories.

10 How do we, sitting here and reading various articles by various persons like yourself, come to any informed judgment as to how accurate or how inaccurate these various dust histories are?

15 A. They're imprecise. I think, in general, where it had been attempted, not only represents a very few -- well, as you know, very few groups -- where it has been attempted, it's probably been done with similar degrees of precision or lack of it. It's dependent upon dust information that doesn't usually go back as far as the cohort goes back, which means that there's some -- actually, the Quebec stuff goes back the furthest, actual measurements of air-borne material.

20 Q. McDonald's work.

25 A. Right. But what that means is that you have to then say -- well, say before 1950, what does the dust information of the 1950's tell us? Well, you end up talking to a lot of people. You find out, or you try to find out, whether or not major or significant dust-control measures have been introduced before measurements were actually made. And the answer, generally, is, no, that very little had been done prior to that time. And that was what we were told. And, as we say in the papers, that's what we assumed, so that ...

30 But, on the other hand, there may have been other factors, which meant that exposures were higher; but what does it mean if exposures were higher? What it means is that we are, in fact, underestimating exposure -- right -- and the dose



5 A. (cont'd.) response curve would be steeper. So, at the very worst, we're being conservative by not saying they were higher than they were in 1950, which I think is the way one should be in public health matters, generally.

But to give you a feel for how good they are, a lot of time and effort went into them; that doesn't mean they're good, necessarily; it's the best we have -- best available information we have.

10 And I am firmly convinced, and I think others who have done this -- the groups that we've already mentioned -- I am firmly convinced that it's better than just looking at length of exposure -- firmly convinced; as are other groups and people who have had a chance to review this information; the British committee, and so forth.

15 Q. Did you encounter any problems even within your own study in terms of measurement, in the sense that different measurements of allegedly the same place may produce wildly different results?

20 A. There's a substantial variation from day to day, from job to job; there just is. People are not exposed to the same level, no matter what they're doing; even if they're on an assembly line, punching holes in a shingle that comes through, every day. They're not exposed to the same thing from day to day.

25 And it's been wisely said that, although people who do work like we do rely on average exposures for a job, to put somebody into an exposure category, people are not exposed to average concentrations; they're exposed to wide fluctuations.

30 I guess the amazing thing is that, when you do this kind of reconstruction, the dose response relationships come out so well. And if they come out so well, maybe there's something to it.



Q. All right; let me turn to one other aspect of dust exposure histories -- or a couple of other aspects.

As I understand it, from reading your articles, in order to come up with the proportions of fibre type in your dust exposure histories, you looked at the percentage in the final product?

A. Product composition; correct -- which isn't ideal. It'd be better to measure each of the fibres in the air, but that hasn't been done by anyone. It's the best we could.

It gives some idea of ... Actually, if you get right down to it, our conclusions are based not so much on quantitative level of chrysotile, say, versus crocidolite, but, ultimately, what we were left with was comparing a relatively pure chrysotile-exposed population with a population exposed to chrysotile and crocidolite. And whether or not that was two per cent crocidolite or five per cent maybe doesn't make so much difference.

I see your point; it's not the best way to do it, but it's the only way to do it at the moment; unless you did some very fancy electron microscopic analysis of fibres collected on a filter, so that you could get different fibre type concentrations. As far as I know, no one's attempted that.

Q. One of the things I thought you had done was, you tried to correlate the amount of silica actually measured with the amount of silica in the final product.

A. No.

Q. No?

A. Not in this study.

Q. All right.

A. No; we measured -- silica ... Well, let me just ... The answer's not entirely "No." Let me just say this; that, in fact, silica air-borne concentrations were measured,





5 A. (cont'd.) just as asbestos in total particles, and where, as much as possible, those concentrations, air-borne concentrations, were taken into account along with product composition in estimating levels of silica exposure; but, in fact, we didn't directly relate air-borne concentrations with product composition.

Q. Did they correlate fairly well?

10 A. I really can't answer that; I haven't looked at that specific question. Dr. Hamad, who is our industrial hygienist, would have to relate -- give you that information.

Q. But I take it, in respect of fibre type, in any event, you didn't because it's so difficult to do, to measure actual different fibre types in air-borne samples?

15 A. Correct.

Q. Can we talk a little bit about conversion to fibre counts.

A. Surely.

20 Q. As I understand it, there's one of your papers, tab 8, where you attempted some conversion factors. I guess if we look at page 492, you've got a table.

A. Right. Actually, I have this table also on this transparency. It may not be necessary to put it up, but ...

MR. McNAMEE: Does everybody have this?

MR. LASKIN: I'm sorry. I'm sorry, Jim.

25 We're on evaluation of dust exposure in asbestos cement manufacturing operations.

30 MR. WARREN: I wonder, Mr. Chairman, if we might make a request of Mr. Laskin -- well, not so much that; just for the convenience of counsel, when you're on that, it would be very useful if we had your order beforehand, because what we end up doing is taking the order in which we've assembled our papers and then try to find the articles which you're referring to, if



MR. WARREN: (cont'd.) you see what I mean.

It would be very useful, I think, for all counsel and for the commissioners as well.

MR. LASKIN: Sorry about that.

Here's what I want to ask you about, Dr. Weill.

Q. When you did your cross-sectional morbidity study, as I read your articles, you applied, for rough purposes -- and I certainly don't hold you to it -- just to see what kind of conversions; there might be a conversion factor of two; is that fair?

THE WITNESS: A. That's correct. That's before we did our own work. Actually, it turns out not to be so bad, but we can talk about that in a minute.

That was before we did this work that resulted in the publication that you're referring to. We were depending on the work by Ayer and Lynch, to which we refer in that paper.

Q. So that, just to use an example ---

A. Yes.

Q. --- you had a dust exposure level in particles expressed as a hundred million particles per cubic foot years, and you applied a conversion factor of two; that would be two hundred fibres per millilitre years?

A. Right.

Q. And if you take an average working lifetime of -- I think you took forty years.

A. Yep.

Q. Let's take forty years. You work out with a fibre standard of five fibres.

A. That's what that would suggest, if all those assumptions are correct.

Q. If ---

A. And then subsequently -- I just want to be sure that the order is correct -- then subsequently it was



5 A. (cont'd.) possible to actually do side-by-side sampling with a midget impinger, and doing fibre counts, which resulted in the publication that you're now referring to.

Q. Am I correct that, if you apply the conversion ratios that you have here, except in respect of corrugated finishing, you would end up with, comparatively, a lower fibre standard for all of these various dust zones, apart from corrugated finishing?

10 A. That's correct. So we would probably today revise that ratio downwards some -- not one, but something between one and two.

15 Q. Does that then suggest that, to the extent that you might find excess risk of disease at a hundred million particles per cubic foot years, and to the extent that these conversion factors are relatively accurate, then expressed in terms of fibres your excess risk is going to result at a lower fibre level than, say, five?

A. Lower than five, I would say, yes.

20 Q. Would it be fair ... Can I ask you how confident you yourself are in these various conversion ratios?

A. About as confident as I am in the reconstruction exposures generally; I think there is validity to that. There is a range.

25 If you took all the studies -- and they're not that many; by the very expression, I suppose I'm suggesting that there are many -- if you took the studies where conversion data have been looked at -- and that would include Graham Gibbs from Montreal, Ayer and Lynch that I mentioned, who did their work with the public health service some years ago; our work, Dr. Hamad in our group, and some information that Dr. Dement has not published yet but has used, from his own use of the environmental data from the asbestos textile plant that he studied,

30





5 A. (cont'd.) there's some information about -- not concurrent; not simultaneous, exactly, but close time periods where fibre counts and midget impinger measurements were made.

I guess that's basically it; that's the body of information on fibre counts vis-a-vis particle counts.

10 Now, if you took all of those, you get a range of ratios, conversion ratios, in a way that gives you sort of upper and lower bounds of risk, doesn't it, in a sense? So I have -- I have some confidence in these conversions.

We give, in that table that you refer to correlation coefficients, and that gives you some idea of how much confidence you should have. As you'll see, the correlation is markedly different for different locations.

15 For instance, if you ask me in the mixing area -- you know, that was the part where the guy was unloading a bag of chrysotile asbestos -- the relationship between particles and fibres has a correlation coefficient of point nine one; that's excellent.

20 Q. Can you, for ---

A. I have a lot of confidence in that.

Q. What is the ... Can you just explain, in simple terms; what does that mean when there's a correlation coefficient of point nine one?

25 A. Well, the higher the ... It means, how well do one set of data correlate, in a linear way, with another set of data; and the higher the coefficient, the better that that relationship is.

Now, at what level can you have confidence is an impossible question to answer, because it depends on what use you're making of the data, and a lot of other things.

30 Some people will say, for instance, that a



5 A. (cont'd.) correlation coefficient of point six is pure guessing and other people will say, "Well, that's not a bad correlation"; and, for many things, it isn't a bad correlation.

But point nine, I think, in anybody's estimate, would be a good one.

10 Q. If you were going to make the conversion from your own studies, would you, ideally, then -- would you have to go back and reconstruct your exposure histories in terms of these differing ratios for every different zone, or would you simply apply your ultimate mean figure, which I take it is one point four?

15 A. The former; and, in fact, you've anticipated some of the work that is going on in our unit at this moment. We are hoping to develop some additional information for all of these dose response relationships that I've demonstrated for you today; some additional information which will at least give some bounds for what this may mean in terms of fibre level exposures.

20 Q. How far are you along in that? Not far enough along to ---

A. Probably not to meet your needs, Mr. Laskin; I'm sorry.

Q. Oh; that's all right.

A. Although, perhaps within the next year, we'll have some of that information.

25 Q. Can I turn just to a couple of points on some of your conclusions from your morbidity study; and I take it, one of the conclusions you came to was that the -- one of the conclusions from your cross-sectional study was that the physiological patterns that emerge from the test that you did supported the theory that these irregular opacities were indicative of asbestosis.

30



Q. (cont'd.) Can you help me as to why, medically, that -- or biologically -- that should be?

5 A. Why the irregular or linear densities are more important in terms of functional consequences than rounded opacities; is that ---

Q. Well, as I understand it, you suggested that the rounded opacities were indicative of exposure to silica and silicosis, and the irregular opacities were indicative of exposure to asbestos and asbestosis.

And I suppose my question, in my own layman's terms, is why should that be?

A. Okay; that's a good question. Let me see if I can answer it.

15 In industrial exposures, where the fibrogenic dust to which the worker was exposed is relatively pure -- that is, those exposures where asbestos is the only known fibrogenic dust in the atmosphere or those where silica is predominantly or primarily the only fibrogenic dust known -- the two radiographic outcomes that you mention are, in fact, those that are seen.

20 Silicosis, in its early stages, called simple modular silicosis, radiographically produces small rounded opacities throughout the lungs. And if you took some lung and examined it under the microscope, you'd see focal silicotic nodules; fibrotic, but hyalinized rounded focal nodules.

25 In asbestos textile manufacturing, for instance, in other exposures, perhaps friction material manufacturing, to some extent, in some areas of mining -- mining gets complicated because there may be this silica around, too, and then again you may get some mixture of exposures; but where the exposures have been relatively pure, one would expect asbestos to produce primarily small irregular many opacities; silica to produce the other.





5 A. (cont'd.) As a matter of fact, it's why the ILO and UC(?) classification, in 1971, added small irregular opacities to the classification, to deal with asbestos-related lung effects; okay?

10 Now, in addition to that, in an earlier study, we found -- and I think it's in here; it's the preliminary -- first paper on X-ray lung function changes -- we found that, if you divided the population up by whether they had predominantly irregular or rounded opacities, the functional consequences were much more substantial for the irregular than rounded. That also is consistent with what we know about what the two diseases do.

15 Small rounded opacities, due to simple modular silicosis, very often will be associated without any functional impairment whatsoever; whereas the irregular opacities as characteristic of pulmonary fibrosis of a more diffuse type, even early on, usually are associated with some functional thing.

DR. UFFEN: Could I ask a question?

MR. LASKIN: Absolutely; sure.

20 DR. UFFEN: When the medical profession refers to silicosis, is the definition one of physical size or chemical composition?

Like, when you refer to silica, you don't just mean SI; you mean silicon oxide or ---

25 THE WITNESS: That's correct. We don't mean just silicon dioxide; we mean silicon dioxide, which appears in crystalline -- free crystalline form.

30 In general, I suppose, when that has to be distinguished from silicates, which of course also includes -- which may be fibrous or non-fibrous, and, importantly, has to be distinguished from amorphous or non-crystalline silica.

This was a very important question for people who



5 THE WITNESS: (cont'd.) got sort of confused about this immediately after Mount St. Helen's erupted and there were some very scary things -- I'm sorry; this diversion -- very scary things in the newspapers about sixty per cent silica and so forth. Well, what in fact most of that was was glass, was amorphous silica.

10 And one of my roles, as a consultant out there, following that time, was to cool everybody down. In fact, only about five per cent of the dust was crystalline silica; part of it quartz, and part of it cristobalite.

15 So we're referring -- when we talk about silica producing silicosis, we're talking about free crystalline silica, and other characteristics that determine silicosis risk, which would include particle-size distribution, and so forth.

MR. LASKIN: Q. Just following up on Dr. Uffen, what then -- what is asbestosis? Is there a medically acceptable definition of what ultimately constitutes asbestosis?

20 THE WITNESS: A. Well, I'm not being facetious by saying that asbestosis is pulmonary fibrosis caused by the inhalation of asbestos fibrous dust.

Your next question will be -- and I'll ask it for you -- how do you make the determination of the latter?

MR. LASKIN: That's my question.

25 THE WITNESS: Well, let me tell you how the committee on which I am the token non-pathologist, which has been formed by NIOSH to deal with the pathology of asbestos-related disease, and to which you referred in your introductory comments; how that committee dealt with it, simply by saying that, in the presence of an appropriate exposure history, pulmonary fibrosis, with one or more asbestos bodies in the lung tissue, is sufficient.

30 Q. In other words, so that the approach was to



5 Q. (cont'd.) look at an exposure history rather than to look at the extent of irregular opacities, or the extent of reduction in lung function or ---

A. From a practical standpoint, since we very rarely have the lung tissue, you might well ask (and I'd be glad to tell you) how I make the diagnosis of asbestosis.

10 I make the diagnosis by eliciting an appropriate exposure history, by seeing irregular or linear densities on the X-ray that meet some minimal level of profusion.

15 I hope it will come as no surprise to any of you that reading an X-ray is not an absolute science that comes out "Yes" or "No." At the very low level of X-ray abnormalities, there's a grey zone -- actually a grey zone on the X-ray and a grey zone conceptually.

20 Some people will read films in that level as positive and some people will read them as negative. I happen to be a fair -- a bit of an over-reader; I tend to read at a pretty low level of abnormality, and people fall -- and this is non-political -- people just fall into -- usually non-political ... People fall into these various categories of readers.

25 As a matter of fact, if you have a series of readers, the computer can account for systematic over-reading or systematic under-reading.

30 What I am saying is that at whatever my level is, "Yes, these are abnormal shadows." They're not just what you might see in a seventy-year-old person with emphysema, who may have one or two little lines; they're diffuse enough to suggest pneumoconiosis. That's enough for me to make a diagnosis; and I do, very frequently.

Q. But it becomes, ultimately, I take it, a judgment call on the part of the person reading the X-ray?

A. That's correct.





5 Q. And what ... Do you have any guidelines that you yourself apply in terms of what sort of exposure history you're looking for?

A. Well, again, I now know, and didn't always know -- I now know that there is a wide variety of exposures that will produce a sufficient dose for this consequence to be possible.

10 That means that there will have been some short exposures, but very high exposures.

15 Many years ago, I showed you an X-ray of somebody who was exposed only for a couple of years -- I'm not sure I mentioned the length of exposure; that was only a couple of years -- but massive exposures at that time, in totally uncontrolled conditions, well, that was enough. Maybe those very high-level exposures for a short time were more hazardous than the same cumulative exposure, but obtained by low level for a long period of time, because of some of the possibilities that I also mentioned, like overwhelming defence mechanisms, and so forth.

20 So I tend to be relatively -- now, I do say that one is likely to have that exposure in the course of an occupation; I've found it in a wide variety of occupations: brake lining, brake mechanics, shipboard marine engineers alluded to, and others.

25 Q. Another conclusion you came to, one you've already mentioned, is, of course, that the dose response relationship and dose being measured by cumulative dose -- can I ask you, if cumulative dose is important, is the length of time that fibres may stay in the lung -- does that have any significance?

30 A. Certainly, its significance can be based on the biological plausibility that -- it has some importance.



5 A. (cont'd.) The problem is, how do you quantify that importance? And you'll be hearing about that later in the week, because there has been an attempt made, as you know, by Geoffrey Berry to deal with this question of residence time.

Is a fibre that was deposited **thirty** years ago more important today than one that was deposited two years ago?

10 I think all of us, knowing what we know about asbestos-related diseases and latency periods, would say that probably it is. I would say that. But nobody has come up with -- and I would say that Geoffrey's paper and his public comments about this would support that; a confident way of modeling that factor.

15 Somehow, it seems reasonable to put additional weight on those exposures years ago.

Q. He attempts some kind of timing with average ---

20 A. Yes, indeed; and he'll undoubtedly tell you about it. But none of his models, none of his sets of data, seem to work totally to his satisfaction, or to the satisfaction of some others. Maybe at some point we'll be able to do that better than we're doing now.

25 We are, in fact, at the moment, looking at that in the animal model and hopefully we'll come up with some information which will allow quantification. We're exposing different sets of animals to cumulative exposure obtained in different ways (high-level exposures for short time; moderate level for longer time) to see just what the difference in biologic outcome is.

We hope that that'll be a good way to do it, but we won't have that information for you any time soon, either.

30 Q. Perhaps, as an outgrowth of that, Dr. Weill, can you help me with how the lung reacts to fibres that get into



5 Q. (cont'd.) the lung; I mean, is there -- I take it there's some kind of clearance mechanism for fibres. Can you talk a little bit about that.

A. I have one very pretty slide; just one single one. Can I show it?

[Some inaudible discussion.]

10 Okay. See that sort of lumpy-looking cell. That's a macrophage, and it is engulfed -- the two ends of that asbestos fibre. This is a scanning electron micrograph given to me by Dr. Brodie, and this long fibre is being -- like all particles that go into the lung, this is being engulfed.

15 Now, that engulfing process is the first step in its ultimate transport away from the lung. It can be transported either up the ... [inaudible] It can either then go up the mucociliary escalator; that is, little projections in the airways and mucus allow it to be moved upward and finally swallowed, or the macrophage can go through the lymphatics, the draining lymph nodes, and away from the lung in that sense.

20 Now, when the macrophage engulfs a fibre totally, as it's not able to do here, that may be better; it may be that it deactivates pathogenicity, or eliminates pathogenicity better than when it tries to gobble up a fibre that's too big, which seems to be the situation here. We're not sure.

25 But, ultimately, what happens is that somehow the presence of these fibres produces the release in these scavenger cells, or macrophages, of certain intracellular substances (enzymes), which, in turn, turn on different kinds of cells, [inaudible], so it's a cellular process.

30 The first step is to try to protect the lungs from that process. The macrophage is very important, and the second thing, I suppose, in a sense, that happens is damage takes place; there's leakage of these substances, which leads to





A. (cont'd.) fibrosis.

5 I guess it makes it clear, even to someone who's not expert at deposition and clearance mechanisms, which includes me, that if, in fact, you had such a high exposure in a peak situation that there weren't enough cells to do the job, or, for instance -- although you have a tremendous outpouring or there wasn't enough room on the mucociliary escalator, or there weren't enough other defence opportunities available that  
10 the cell that has not been deactivated has better -- I mean, a fibre that has not been deactivated because of this overwhelming will produce more disease; at least, that's a possibility.

But, again, you've got a local expert here that can perhaps expand on that or correct it, as the case may be.

15 Q. Is it fair to say that the more able the lung is to clear the fibre from it then the less dangerous the exposure is going to be? Is that a fair conclusion?

A. Well, I think it's fair to say that with hazardous particulates in general, including fibrous particulates, including asbestos fibres, that the more effective the clearance mechanism, the lower the risk of disease.  
20

Q. And from what -- a comment you just made earlier, the ability of the -- is it fair to say that the ability of the lung to clear the fibres may depend on the intensity of the dose at a particular time?

25 A. I think it could. That's speculative at this stage, but I think it could.

There's a bit of evidence that I, again -- others may have -- there's a bit of evidence on that point, but I don't feel comfortable -- animal evidence -- feel comfortable in detailing it.

30 Q. I take it there is some evidence from some people that an intense exposure in a short period of time may



Q. (cont'd.) carry with it greater excess risk than the same exposure over a longer period of time?

5 A. Enterline's work seems to suggest that; ours may suggest it, but it's possibly confounded by the fact that those people also had blue asbestos exposure.

Q. But even in your work, there seems to be the maintenance workers who got the intermittent high exposure had a higher excess risk.

10 A. That's correct; yes, sir.

Q. What about in terms of lung clearance, does fibre size play a role? I take it it does, from what you said.

15 A. Yeah. I think the shorter fibres are more likely to be totally engulfed and handled by macrophages in the long run, which has led some people to suggest that that may be the mechanism, while longer fibres, in most quarters, thought to be more hazardous than shorter fibres.

Q. Is that the current thinking; that the longer the fibre, the likelier it is to be more hazardous?

20 A. Well, there may be a limit, but I think most people would say that fibres longer than eight to ten micron in length are more hazardous than those shorter.

25 And this has been found with inhalation studies, intrapleural installation studies. I think that most people would --intrapracheal injectin studies. I don't know of very much contrary evidence, but that doesn't necessarily mean there'd be total unanimity of opinion; I think that's the general view.

Q. Does the ability of the lung to clear the fibres depend upon the age of the particular person?

A. I don't know.

30 Q. I guess, ultimately what -- I mean, one of the things this Commission is concerned about is the susceptibility



Q. (cont'd.) of children.

A. Yes, of course.

5 Q. Is there any evidence, or is there any work in that area that would suggest any conclusions about the susceptibility of the lungs of young children as compared to adults?

A. I'm not aware of any relevant to this sort of question. Again, I would refer you to one of your eminent local scientists (Dr. Murer), who may have some information on that.

10 Q. You yourself have no opinion on that, one way or the other?

A. No, sir.

15 Q. Let's turn to another conclusion of yours, which I think you touched on in your opening talk. As I understand, one of the things you said in your article at tab 4, which is lung function, consequence of exposure in asbestos cement product manufacturing plants, is that a person can have asbestosis and, to put it simply, no change in the X-ray, or no significant change in the X-rays and, conversely, a person can have asbestosis and no significant reduction in lung function.

20 A. By the way, you've reminded me about something which I didn't make clear. I was concentrating on X-ray and lung function change with asbestosis versus the cancer risk, and I didn't say something else that I think you are saying now, and I should have said; and that is that, in the population in general, in our population generally, the X-ray and lung function  
25 were more or less equally sensitive in picking up a dose-related response.

30 Having said that -- because the elbow in the dose response curve was at the same level of exposure -- but having said that, there will be individuals who will have functional evidence first and others who will have X-ray evidence first.

Q. Is the conclusion from that, if you're talking





5 Q. (cont'd.) in terms of the kinds of medical examinations, from a medical point of view, that an asbestos employee should have, that he should have both of those?

A. Yes; that's my opinion.

Q. And at what kinds of intervals?

10 A. All right. Asbestosis is a slowly progressive disease; progressive, no doubt, but slowly progressive. This is particularly true now as opposed to the days of Montague Norry, around the turn of the century.

15 My own feeling is that annual examinations are too frequent and not necessary. We've now looked at many, many films taken at annual intervals, and it is extremely unusual to be able to discern a change in a twelve-month period due to asbestosis -- now, something else may occur, but due to asbestosis.

20 And, as a matter of fact, in a longitudinal cohort, the interval is three years, and I think that's not a bad interval. Now, somebody might say two years; okay. Annually is too frequent and not necessary, and maybe the cost benefit analysis might suggest even though the risk of the examination is very, very small, the benefit is possibly nil.

So that I would say, something less frequent than annual, and maybe three years would be sufficient.

Now, the cancer question, of course, is related to that also, but I don't know if you want to ...

25 Q. Sure; now that we're on it, let's talk about that.

A. The question is, does it do much good to take annual film in the hope of detecting a neoplasm at a time when it's treatable? That's the question.

30 And the answer is that, where that has been studied, the results are not very encouraging. In studies of lung



5 A. (cont'd.) cancer surveillance (in Philadelphia or in the Mayo Clinic I think are probably the two major ones), the results suggest, even in high-risk groups, that the frequent X-raying of these individuals doesn't really have a major impact on longevity. That's sad, but seems to be true at the moment.

Q. Why is that; because it's not a treatable disease?

10 A. Not treatable at the stage that it's diagnosed, usually.

Q. I see.

15 A. Not invariably true; that doesn't mean that all lung cancer has a fatal outcome; but frequent monitoring, at the moment, doesn't seem to have shown substantial gains in cure rates.

Q. Is that something that you yourself have found in your own practice?

A. We haven't looked at that question.

20 Q. Just one final question about your cross-sectional study, and it goes back to fibre typing: is there something about crocidolite, from a medical point of view or from a chemistry point of view, that would tend to suggest that it is a more dangerous fibre than chrysotile?

25 A. Well, the thing that most people have latched onto, in terms of an explanation, is, it is a straight, thin fibre as opposed to being more or less curly fibre; chrysotile isn't quite as curly always as the pictures suggest, but it does tend to have that configuration.

? 30 The straight fibre is at least possibly likely to penetrate more deeply; that's one aspect. There's now good evidence that chrysotile fibres in the lungs do not have the same long-term history as amphibole fibres, including crocidolite.



5 A. (cont'd.) Chrysotile tends to be broken down; magnesium can be leached and isn't generally thought to be as persistent in the lung tissue as crocidolite. That seems to me to be another possible reason for the differences in pathogenicity.

10 First, is it deposited more -- is it more likely to be deposited peripherally because of its physical characteristics, dimension; and then, second, is it likely to be retained longer because of its perhaps chemical composition?

If the answers to those questions are "Yes," that certainly would give us a clue concerning the differences in pathogenetic potency.

15 Q. Can I turn to your mortality study.

A. Yes, sir; what tab is that?

15 Q. And could I just ask you about this ---

A. I'm sorry; what tab?

20 Q. Am I correct that -- you've got two articles; the first at tab 7, which is influence of dose and fibre type, and the second at tab 11, lung cancer risk associated with manufacture of asbestos cement products.

Do they essentially involve the same conclusions?

A. One was presented at the Leone meeting on inhaled fibres, and the other was published in the American Review of Respiratory Diseases.

25 Yeah; there's minor differences in what data are presented, but it's the same information.

Q. Can I just ask you about this tracing problem; can we discuss that for a minute.

30 As I understand it, you went to Social Security, and of the cohort that you studied, you were able to ascertain from Social Security records that eleven per cent had died, sixty-four per cent were still alive, and twenty-five per cent





5 Q. (cont'd.) you didn't know about one way or the other, and you couldn't isolate who the twenty-five per cent were because of confidentiality regulations of the administration.

A. At that time.

Q. At that time.

A. That's correct.

Q. Which have now changed.

10 A. Yes.

Q. Do you get any assistance from, for example, determining who applies for death benefits; I mean ---

15 A. That part of it was very favourable in terms of supporting the assumption that most of those who had not applied were in fact alive; because, in the States, not only is a survivor entitled to apply for death benefits, but a funeral director is entitled to apply for death benefits.

So it is to the interests of the mortician to notify Social Security of a death since, if he does so, he gets paid for that particular funeral.

20 There's every reason to believe -- some of this is in the paper -- there's every reason to believe that you don't miss a lot of deaths that way.

25 It is interesting that the present administration, the most recent one, is suggesting as one of their cost-cutting -- Social Security cost-cutting measures, to eliminate the provision that, if there are no survivors, a death benefit is paid to the mortician, which is going to make it harder for epidemiologists, but I'm sure the administration doesn't really have epidemiologists in mind when they've suggested that. That is one of the provisions that has just been suggested. When I read about that, I wasn't all that happy about that.

30 Well, getting back to the point, so that not only



5 A. (cont'd.) survivors, but others can, in fact, apply. So if somebody is buried, dies and is buried, the odds are very high -- in that steelworker study that we suggested -- very high -- but it doesn't eliminate the possibility that there is some under-reporting of mortality, and we clearly say that.

10 Q. I suppose, for example, because some people could leave the country?

15 A. That's correct. I mean, they're all -- you know, you could conjure up, certainly, some scenarios that would fit. Well, people who aren't covered by Social Security in our country, and there are such groups as well.

20 Q. Are they -- are those groups likely to appear in employed or formerly employed workers?

25 A. They might, because they could leave this industry which is, of course, covered by Social Security, and go to an industry that wasn't.

30 Q. This industry is covered by Social Security?

35 A. Oh, yes; totally. But the V.A., Veterans' Administration, for instance, would not be -- or was not at that time. I think it is now, actually.

40 But I guess that, in addition to that kind of argument, the major support for the validity not being affected by this trace problem is pretty much what I tried, and this takes a little time to go into.

45 There was a, as you might expect -- you know, in that low-exposure group there were a lot of these short-term employees. You have to remember, again, that sixty per cent of this cohort had less than one year employment, much of that a long time ago.

50 Now, they will have gone -- there's a lot of emigration out of the south, particularly among blacks; so they



5 A. (cont'd.) would have gone to different places and hard to trace. So the trace was not equally distributed among the exposure groups. Among the highest exposure groups, which also meant the longest-term employees, the trace was best.

10 If you say that the overall mortality rates, which tended to be what you'd expect (one or a hundred in those groups), was really what the others should have been, and we're willing to say, as a worse-case situation, that in fact was possible.

15 Well, let's raise the overall mortality in those groups up to where ought to be, assuming that we missed some deaths there, and also raise the lung cancer deaths by about the same proportion, okay, we still get to a totally overlapping situation in those low-exposure groups of overall mortality and lung cancer mortality, suggesting to us there isn't any increased risk of lung cancer mortality in the lowest-exposure groups, which is consistent with some other studies, as you know.

20 Now, the only fly in the ointment would be is that for some reason the likelihood of tracing somebody was related to cause specific mortality, as I said, and I just can't conjure up, after months and years of thinking of it (and many other people in my group -- statisticians), why that ought to be; why should there be any relationship between cause specific mortality and trace.

25 And if you will agree that there shouldn't be, and there's no reason to assume there is, even in a worse-case situation, then I think one is left with the conclusion that these are valid.

30 Now, the final judgment on that will almost certainly have to await this updating of our study, and ---

Q. Well, when you say there should be no





5 Q. (cont'd.) relationship between trace and cause specific mortality, I mean, do you mean by that that this twenty-five per cent that you haven't located should have a mortality that's random with the ---

A. Similarly distributed; yeah.

Q. As what; the general population or the population of workers? The population of workers?

10 A. Exactly. So that those who were lost in the high-exposure groups should have a -- you know, a relative risk of three times the expected in the lowest; perhaps no. That's what I mean.

MR. LASKIN: Maybe, Mr. Chairman, a convenient point to break for lunch, in view of the hour.

15 DR. DUPRE: Will you be continuing with your examination?

MR. LASKIN: For just a little while, Mr. Chairman; I should have about another half-hour.

20 DR. DUPRE: Now, I might perhaps just ask the parties to roll over the following in their minds over lunch: as I understand it, counsel, we can only meet beginning at 11:00 a.m. tomorrow morning because of time commitments, and we will have only about two or three hours, at most, available for testimony tomorrow. Is that correct? We have the balance of this afternoon.

25 MR. LASKIN: We certainly have the balance of this afternoon; you're quite correct. For reasons of time commitments, we have to start at 11:00; and leaving aside for a moment Dr. Weill's own commitments, I have a personal commitment in the afternoon, but that doesn't necessarily mean that we need stop. I suppose those are the parameters

30 DR. DUPRE: Dr. Weill, of course, permitting, the Commission would be open to some time this evening, so let me



DR. DUPRE: (cont'd.) just leave all of this with the parties.

5 Dr. Weill, if it is necessary, would you be able to spare us an hour and a half or so after dinner?

DR. WEILL: Of course.

10 DR. DUPRE: So may I leave this with the parties, and perhaps, by the time you finish your examination around 2:30 or so, the parties can give me some kind of an idea of how they would like to plan the geography of the rest of Dr. Weill's testimony.

MR. LASKIN: Are we adjourning -- till what time, Mr. Chairman?

DR. DUPRE: Shall we adjourn until 2:30?

15 MR. WARREN: I guess, if I had a preference, to state it, it might be that if we could finish today, which is I think implicit in what you're saying about this evening, I would certainly prefer it, since I would rather not stay over if I can avoid it.

20 I guess another thing I might say is, Mr. Laskin, at least insofar as my questions go, is doing an able job of covering a number of things I want to cover, so I think he's going to make it shorter for me, at least.

DR. DUPRE: Well, shall we resume, say, at 2:15, and see what .....

25 INQUIRY RECESSED

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THE FOREGOING WAS PREPARED  
FROM THE TAPED RECORDINGS  
OF THE INQUIRY PROCEEDINGS

30   
DEREK WEST



DR. DUPRE: Doctor, are you ready?

DR. WEILL: I am, sir.

DR. DUPRE: Counsel, will you proceed, please?

MR. LASKIN: Thank you, Mr. Chairman.

MR. LASKIN: Q. Doctor Weill, just one final question on this tracing matter. Did I understand you to say when you were giving your initial talk that you will now be able to, because of the change in confidentiality regulations, you will now be able to deal with this twenty-five percent?

DR. WEILL: A. Yes, to this extent. In that twenty-five percent last time...by the way, a couple of points. Let me back up just a little bit. That twenty-five percent, for instance, today...even under the same regulations...would not be likely to be twenty-five percent, if you understand what I'm saying. It is likely that in different times that proportion of lost-to-view group would be smaller, but still, it would still be very formidable to try to trace everyone who is not known to be dead...which is what we were faced with. We tried it, and as the paper says, we were not very successful and didn't have the resources to do it successfully.

Now this time, we in fact know what proportion is lost to view because they have told us who they are having financial transactions, money going in one direction from Baltimore, or in the other direction. That leaves us this time, because we are well into this analysis or study, data collection phase actually, that leaves us with thirteen hundred people to trace and about two thousand are now dead, which means that the others, the remainder, are people who Social Security have told us cheques are going one way or the other.

So we are in the process of tracing those thirteen hundred people. We've done that and we've already traced several hundred, so that's where it goes.

Q. You had six hundred deaths...





A. No, we had twelve hundred in the total cohort, six hundred if you limit it...which we did...to only those who had had twenty years followup or more.

5 Q. Have you...you are continuing to follow this cohort?

A. It's the same cohort.

Q. The same cohort...

A. Except five years later.

10 Q. Okay. Do you have any figure that corresponds to the six hundred deaths figure?

A. I don't.

Q. Okay. To return to some of your conclusions of your mortality study, can I first of all take you to one of your two articles, the first one of which is at tab seven, and the table two at page 348.

15 A. Okay.

Q. Have you fitted any of your data, can I ask you, to any dose-response relationships, any curves or...?

A. Yes, sir.

Q. Can you...what can you tell us about that?

20 A. Okay. Thank you for that question, because I think it is a matter that has relevance to what public policy needs to consider, or people who make public policy need to consider.

25 There has been a lot of discussion...if you look at this figure, which comes from the paper, and we've drawn some lines along there...forget the lines for the moment and remember that there is for each of these observations a range dealing with the ninety-five percent probability confidence interval, which of course means that at that level of confidence the point may be anywhere in between to out of bounds.

30 Now, most of the time when people who have looked at the shape of the curve recently, most of the time



5 DR. WEILL: (cont'd.) that they are in fact  
expressing an opinion, their opinion will be couched in the  
sort of language - it would be that there is nothing about  
our data that precludes or excludes a linear dose-response  
curve for carcinogenesis. That certainly is a statement that  
I could make with these data as well, and if you look at the  
linear curve or line, slope, here you can see that it is not  
only linear, but usually the implication of a lineal line is  
that it goes through the origin, that is once you get beyond  
10 zero exposure there is some increase in risk. That's bio-  
logically plausible, the evidence for that to support it  
is not conclusive, I agree with that, but certainly there is  
nothing to suggest that it isn't linear, and this line going  
through the origin in our data certainly goes through all  
15 the points that envelope the ninety-five percent confidence  
interval for each observation, or average observation of  
deaths.

On the other hand, there are other lines  
that could be drawn to these tables, and this is true of  
other peoples' data as well. Let me give you a couple of  
20 for instances, without necessarily saying that any of the other  
shapes are the correct shapes. I am just saying there are other  
possibilities.

Let's look first at the cumulative normal line  
which I think...where's my statistical colleague...can also be  
called the logistic line, is that about right?  
25

Anyway, it's called a generally cumulative  
normal line. This was drawn for me by Dr. Hughes, who is  
the statistician on our mortality work, and you can see what  
happens here, is that it's a line which is very flat at the  
beginning, the low exposure level, and then it goes up and then  
again flattens out after some large, greater level of exposure.  
30

Now if this line also says that for every bit



5 A. (cont'd.) of exposure after no exposure, after zero exposure, there is some increase in risk. You can't see it. I mean, it's not drawn that way, but there is some... mathematically a line would be constructed in such a way that there would be some increase in risk.

10 From a practical standpoint, I suppose, one can question several things. Down here, is that risk ever detectable, and then the second question which is perhaps even the more difficult one is, if it's not detectable and it's only theoretical, is it a risk which is acceptable. That obviously is a question that must be answered by people other than just scientists. It's a public question that requires a broad input.

15 Someone might say that a logistic..and this again, this line constructed passes through all the ninety-five percent intervals...possibly one might say that this in effect is a threshold line that below some point here there isn't any risk that we could ever measure, and therefore perhaps it's a risk that are similar to risks that we take in many other walks of life. That's not necessarily what I am suggesting is my opinion, but it is certainly an opinion which might evolve.

20 Then finally, there is a true threshold, that linear dose-response relationship, which is the dashed line, which says that at some level of exposure, let's say this level, there is zero risk, no risk...and that that risk only increases beyond that.

25 Well, our data would be consistent with that line as well.

30 Now, I don't know whether I've uncomplicated or complicated the situation, but I hope I have done at least one thing...that is, indicate to you that estimation of the shape of the dose-response curve in that very low part of the





5 A. (cont'd.) curve, that low-dose part of the curve, is very difficult, that because of the methodology available to us there is going to be uncertainty, and in this case we call that uncertainty 'confidence limits', and then you have to decide what the most likely part...most likely shape is.

10 It certainly seems to be clear to me that you have data up here in this range...and we all have data in this range up here...and just because that relationship is relatively linear, and many people have shown that, the Quebec study, Enterline, and so forth, that doesn't necessarily mean as this cumulative normal curve shows, that is relatively linear here, it doesn't necessarily mean that it is linear to the intercept.

15 I'm sure there will be some comments about that, or questions.

MR. LASKIN: Q. I take it one of the things that having a linear curve throughout does for you, is that, if you will, it is a more conservative estimate of risk and it provides some margin of safety, I take it?

20 THE WITNESS: A. Yes, the most conservative estimate, the shape of the curve.

Q. Is that one of the compelling reasons...

25 A. Well, I mean, that's the most conservative. I suppose even a more conservative one is, our data won't fit this, and that is to say there is a convexity upward, that it takes a very steep increase at the low end of the curve, which would overestimate or at least make the risk appear even greater per unit dose at the low end than later. Do you follow...?

Q. A kind of reverse thrust?

30 A. Yes, that's it, right. So it isn't the most conservative shape that you can imagine, but of these three it is the most conservative.



Q. Is that one of the reasons why numerous people postulate a linear dose-response curve?

5 A. Counsel, that's a hard question. That goes to motivation. I would say that that probably is a reason.

Q. Well, then, I'll pin you down as a statistician, but can you explain for people here what a cumulative normal curve is? Explain for me?

It's some kind of logarithmic calculation?

10 A. Well, it's a shape...I guess the hearings don't provide for someone in the audience that could do this better than I, to explain it, or do they?

Q. Well, ultimately.

15 A. Ultimately. All right. I just...I mean it has an S shape of this sort. In fact it has...I think a lot of proportions tend to have this kind of shape, but there are... in fact there is a small increase at the low end, and again a small increase at the high end, and at the greatest change per unit, whatever is on the other axis, is in the middle, and it has a sigmoidal shape.

20 Now, that's statistically not a very satisfactory explanation, I'm sure. That's as much as I can tell you.

Q. You had a slide up earlier in your original presentation where I believe you showed some kinds of dose-response relationships which weren't as smooth, which kind of had a jag to them?

25 A. Sure, and that's the nature of the beast, because all of our points are derived from imperfect data, and that's points dealing both with exposure and in biologic response.

As a matter of fact, quite from being disappointed in how they sometimes had a...what did you say, jag?

30 Q. I think you called them elbows yourself.

A. Elbows...quite apart from disappointment



5 A. (cont'd.) stemming from that, I am always amazed, in fact, they tend to look pretty good. Well, one way of explaining the elbows is that below that level there is no effect.

I don't think we have...I really think it is, at this point in time, unfair to say...am I allowed to do this?

Q. Sure.

10 A. Yeah, the curve is something like this and this is the so-called elbow. I don't think at this point it is fair to say that the possibility at this low level of exposure there is no effect. I mean that certainly is a possibility, if you wanted the alternative.

15 Another alternative is that we can't detect the effect. That it's there, but our measurement tools are not sensitive enough to allow us to detect the effect.

20 One of the possibilities is that, yes, indeed, as terrible as this sounds in some quarters politically, that there is a level of exposure that does not in fact lead to some adverse health outcome, and I think we have to accept that as a possibility.

25 After all, we are all exposed to radiation. Theoretically not everybody has adverse effects from that irradiation...or maybe we can't detect it.

30 Q. Dealing only with the industry that you have studied, the asbestos cement products, are you prepared to venture any professional opinion as to whether there is that, I suppose, threshold below which there is no excess risk?

A. I am prepared to say that the risk at levels below what we have measured, or estimated, to be a hundred million particles per cubic foot years, the response is very small.

35 On the basis of our studies of morbidity and mortality to date, it's a risk we can't detect.





A. (cont'd.) Of course as our work continues, we'll be able to amplify on that, confirm it or refute it, and we won't be afraid to do the latter if the data don't support it.

5 Q. Could we, after the hearing, could we have the liberty of making at least a copy of that document?

A. I've got the paper on all these and I'll have them copied for you and return them...or I can even do them here, if you want.

10 MR. LASKIN: Can I.....I know Dr. Uffen has a question by way of followup...can we, just for the record, make that exhibit number eight, which is, I take it, the dose-response curves that you have fitted to your own data.

15 EXHIBIT # 8: The abovementioned document was then produced and marked.

20 DR. UFFEN: Mr. Chairman, just as a more generic suggestion on this score, I think it might be useful when we have slide shows such as this, if we have new slides, generally to make them exhibits, because I think it will make a cleaner record. When a witness is talking from a slide or a diagram, it's awfully hard to reconstruct it once you go back and read the transcript, unless there is a document to refer to. I think a lot will get lost to posterity or whatever, if we don't have some record.

25 MR. WARREN: Mr. Chairman, just as a matter of information, on my presentation all of the charts and figures do in fact come from published articles. This was the first thing that I showed today, that does not.

30 MR. LASKIN: In response to Mr. Warren, because I think it's a very valid point and it's one Miss Kahn and I tried to anticipate, there is a difficulty in reproducing slides in a short period of time if the particular person who brings



MR. LASKIN: (cont'd.) the slides isn't in a position to leave them.

5 With respect to Dr. Weill, because of the very point that he made that all of his slides were already reproduced in his articles, it didn't seem to me to be necessary for this witness.

But to the extent that we can, yes, we will try to accommodate the Commission.

10 MR. WARREN: Again, I don't want to be procedurally...I think that the sentiments you are expressing, I agree with a hundred percent. We should do the best that we can here. This is not a formal trial or anything like that, but I do think from everybody's standpoint it is better to have the document to refer to in the future, when we are going back and looking at transcripts.

15 So I think we are saying the same thing. We would like to see this one as an exhibit.

Dr. Uffen?

20 DR. UFFEN: Just a little followup on this exercise. I think it was about the third or the fourth slide you showed this morning, it was blue and white, it had three sections on it and down at the bottom there was sort of a total.

I was curious...if I remember it correctly, it actually showed a negative. Not just sigmoid-shaped curve, but your actual data had the curve. It would appear to be strange...

25 DR. WEILL: No, it isn't strange.

DR. UFFEN: Is it real?

DR. WEILL: It was real. You are quite correct. That is exactly what it did show, and it's not at all unusual...

DR. UFFEN: The increased dose...the lower response?

30 DR. WEILL: ...for the highest exposure...yeah, because there is a survivor effect.



MR. LASKIN: Can we put that on the projector?

DR. UFFEN: That's the one.

DR. WEILL: I think that may show the point.

5 Does it...?

DR. UFFEN: Just at the bottom of the chart. There it is.

DR. WEILL: Yes, that's right. Well, there is a slight...of course, those differences are...you remember, that comes from the same work that has the confidence limits, so obviously the confidence limits of these are going to, you know, be like that.

MR. LASKIN: You are fitting the means there, I take it?

DR. WEILL: These are just the means, that's correct. But nonetheless, I think the point is a good one, that sometimes you do, in your highest exposure group or your longest exposure groups, get a dip down in the...

DR. UFFEN: But what about that second point which is down in the low exposure, and yet it produces a lower response for a higher dose? Is that real?

DR. WEILL: Do you mean this?

DR. UFFEN: The second point from the left. That one.

MR. LASKIN: Just for the record, you are pointing to the chart on Risk of Respiratory Malignancy, and the point at fifty to a hundred degrees, total dust exposure.

DR. WEILL: All I would, again, have to say there, is that the precision with which that measurement can be made has some bounds on it, and that kind of difference is not a significant difference. There is a tremendous overlap in the ninety-five percent, and I don't think that that has any meaning.

DR. UFFEN: That's not the survivor...?





5 DR. WEILL: No, that isn't. I'm sorry, I thought you were referring to the...see how the prevalence of irregular opacities goes down in the last...that is not an uncommon finding in this kind of work.

MR. LASKIN: That may be, Dr. Weill, one slide which we may not be able to find in your articles. I wonder if there is any possibility that we could at least arrange for a copy of that particular slide?

10 DR. WEILL: Sure. I'll tell you what I'll do. I have another one. Can I just hand you this?

MR. LASKIN: Thank you very much.  
Exhibit nine.

15 EXHIBIT # 9: The abovementioned slide was then produced and marked.

MR. LASKIN: We'll get it reproduced and circulate it.

20 DR. WEILL: If I don't have another one, I may have to ask you to send me a copy as well. I think I do.

MR. LASKIN: Can you just, while we're on the point Dr. Uffen brought out about survivor effects, can you just tell us very briefly about that?

25 DR. WEILL: Yes. Both in cross-sectional studies and in longitudinal studies, the finding of disease depends upon the participant of the cohort being there for you to measure disease, or being there for us to measure disease.

30 In certain instances or often for reasons of disease, but they are other reasons, such as, for instance, somebody being transferred out of an area once x-ray changes occur, or whatever reason, the population that you are studying at any given time may have some of the people of greatest interest, not there...either because they have become ill and possibly died



DR. WEILL: (cont'd.) of their disease, or for some other reason as I have mentioned.

5 Now, the survivor effect is a little less likely to be very troublesome in slowly-developing low-latency problems such as asbestosis, because very often people aren't suddenly aware that they have an illness and they leave the industry, although at some point that is certainly likely to occur.

10 As far as malignancy is concerned, that is a problem, and well, it's a problem that you probably have recently heard about in terms of retirees and whether or not at age sixty-five, for instance, some people might already have developed an adverse effect and left the industry, and that sort of thing.

15 So it's a real thing. It's particularly important to a group like mine. We have as large an interest in occupational asthma and airways diseases, and if somebody is aware of wheezing, acute shortness of breath everytime they go into a plant, they are very often likely to recognize that and know it's causal association and leave, so that if we go into a plant manufacturing, say detergent enzymes, and want to find out how many people are affected by sensitization and exposure to this sensitizing agent, we are going to underestimate the risk because some people will have left. That's the survivor effect.

25 MR. LASKIN: Q. While we are on effects, I take it there is another effect that is common in some of these articles, called the health worker effect?

THE WITNESS: A. Yes.

30 Q. Do I take it from that, that to the extent that you are assessing relative risk by using national mortality data, or indeed any data that isn't occupationally related, that you may underestimate the risk on the theory that workers by and large are more healthy than the rest of the population?



5 A. That certainly is a possibility. Well, it's one way that we might account for our low SMR's, standardized mortality ratio, that this working population is in fact healthier than the comparison group.

On the other hand, most people think that the healthy worker effect has some sort of finite end, so that it doesn't last forever, and at some point...ten, fifteen or whatever years...after work has ceased, you should no longer be finding that.

10 Q. Can we turn the page on tab seven to page 349, and right at the end of the first full paragraph on the lefthand side, you indicate the possibility that mesothelioma, the tumor you are referring to, has been underdiagnosed in your study.

15 A. Well, we think it has, yes. Of course, in 1960, attention was first drawn in the literature to exposure to asbestos and this tumor. It took certainly into the mid-sixties, if not the late-sixties, really, for there to be general appreciation...and by general I mean physicians around the country, this continent, as well as western Europe...  
20 really understand that there is an occupationally-induced tumor called mesothelioma.

Now during that period pathologists were not really totally attuned to this diagnosis. It's a difficult diagnosis. It was then, it continues to be. In summing up the asbestos part of the Leone conference, I indicated that  
25 it has been somewhat of a disappointment that even the mesothelioma panels that are now operating in Canada and the United States and in Britain have not really been as helpful in reducing the interobserver variability, the variability in diagnoses between one pathologist and another, in many  
30 instances for this tumor. I think for a long time there has been underdiagnosis. I think some of that has been catching up.





5 A. (cont'd.) I think there is a more general awareness. I think the difficulties are becoming better recognized. There is, of course, a tremendous public interest, and that includes physicians, and that includes pathologists, public interest in asbestos-related health effects, mesothelioma has been talked about quite a bit. Famous people like Steve McQueen get mesothelioma, everybody reads about it in the newspaper.

10 So that there may be not just an appropriate amount of number of tumors diagnosed as mesothelioma now. One might even consider the possibility that there is some overdiagnosis in mesothelioma, and this in fact has been suggested by some of the work of the panels, that have not confirmed as many as fifty percent of the suspected cases submitted to them.

15 Q. If you misdiagnose it, what are you likely to diagnose it as?

20 A. You can go in either direction. You can miss it and consider it a metastatic carcinoma and not recognize it as mesothelioma, or in the other direction the most likely thing that mesothelioma...a tumor diagnosed as mesothelioma...there is not as likely to be...terrible syntax, but I apologize...is an adenocarcinoma, which has a tendency to spread widely along the pleural surface.

25 So there are in fact likely to be a number of cases, particularly people who have been exposed to asbestos, where the diagnosis would be in error.

30 There is one other point. I have already indicated how difficult it is to make the diagnosis. It's difficult enough if you have tissue obtained surgically or by some other biopsy method, quite difficult, and there are those people who believe that the very.....the most credible way, valid way of making a diagnosis is at autopsy when a primary tumor, particularly



5 A. (cont'd.) adenocarcinoma elsewhere, has been totally excluded. Autopsy rates everywhere have been decreasing considerably, in our country, in yours I'm sure, certainly in western Europe, and that, too, may be a factor in relative overdiagnosis recently.

10 It's a possibility. But we think there were more. As a matter of fact, since the cutoff on this study we have been made aware of more mesotheliomas. I'm almost sure that our updated mortality study will show more than two mesotheliomas. Not almost sure, am sure.

15 Q. Does the evidence still suggest, or is the evidence differing as to whether mesothelioma is very cause-specifically related to asbestos exposure?

20 A. I think it's related to asbestos exposure in a high proportion of cases, but not all.

25 Two obvious points come up. One is that there is endemic in a portion of Turkey, the country Turkey, where there is no industry and no asbestos, but mesotheliomas have been present for generations, and perhaps centuries. We've been... I have been involved personally in the WHO, IARC effort to deal with evaluating the fiber effect in this rural population, and recognized by the people there locally in conjunction with the mineralogists in South Wales and in Paris, that there are fibers in the soil and in buildings built with this material. They are called fibrous zeolites and the specific fiber is an erionite fiber, and the mineralogists that deal with fibers in tissue have demonstrated there erionite fibers in the tissue of inhabitants of Kahrane, which is a little town there, who have mesothelioma. So it is a nonasbestos fiber which presumably is etiologically associated with the development of this tumor.

30 Then of course the other bit of evidence is, that no matter how hard one tries, and people do try very hard



5 A. (cont'd.) these days particularly in cases, no matter how hard one tries, in every series there is a varying, but at times substantial, portion of cases of mesothelioma where no occupational, no residential and no domicile area exposure to asbestos has been established.

Q. Which may suggest there may be something...

A. Some other causes.

10 Q. I take it zeolite, for example, we don't have that in North America, but...

A. We do.

Q. We do?

A. As a matter of fact...

DR. UFFEN: Water softeners.

15 DR. WEILL: A. We have fibrous zeolites. In fact we have specifically those erionite fibers. You might be interested to know that in some of the tracks where they want to build the MX missile track system, there are such deposits. It says something for our defence department that they would publish the maps of where we are going to put these tracks... I'm not sure what it says, it's inappropriate for these  
20 hearings, perhaps, but...the track was published, the map, and the geologists, mineralogists, at either Yale or Princeton said, my gosh, they are going to be digging right through some erionite deposits, and actually made this known to government agencies and local people and so forth, and there was a little stir about it. I'm not sure anything was ever done.

25 But the point is, we do have, yes.

Q. But I take it, at least from your readings, that if you've got somebody occupationally exposed to asbestos and that person develops mesothelioma, it's a pretty safe assumption that there is a causal relationship between the two?

30 A. It's my judgement that that tumor should be considered attributable to the exposure in the workplace.





Q. Do you.++.

A. Again, based on probability.

Q. Do you make the same judgement with respect to asbestosis?

A. No. Not simply exposure, because in contrast to mesothelioma, the background of lung cancer in the civilized world is very high, extremely high, and increasing continuously. It accounts for essentially most of the increase of cancer rates in recent times in North America and, I'm sure, in Britain as well.

Now having said that, one would think...and I think most people would agree...that most of that increase is related to smoking. Now, some of those tumors, and probably the number is somewhat in question, a former secretary of Health, Education and Welfare made some estimates and others have not agreed with those estimates..and I won't get into that...but the point really is that the overall impact of lung cancer incidence and mortality in the two countries is going to be only in small part, in my judgement, in the judgement of most people, due to this occupational exposure.

So we are left with the question, well, how do you tell when a lung cancer should be considered attributable to the exposure? For some of the reasons that I've already mentioned, primarily because I think the level of exposure which is associated with this increase is also a level of exposure likely to produce some evidence of fibrosis. It has been my conclusion, and this is a matter of public record and it's in papers and things, and it happens also to be a conclusion...I'm not alone in this...I don't pretend to be. This is also, I think, the scheme used in compensation boards, pneumoconiosis panels in Britain, that for a tumor to be considered attributable...a lung cancer to be considered attributable, there has to be some evidence of asbestosis.



5 A. (contd.) Now, unfortunately that is not a perfect way to make that judgement. I would say almost certainly, because of variability, there will be some unfortunate individuals who have an asbestos-attributable lung cancer who don't have evidence of asbestosis. But the tumor doesn't say, I'm related to asbestos. You have to use some guidelines.

10 The only reasonable guideline, short of counting every lung cancer in a smoker who has also been exposed to asbestos attributable, short of doing that...and I think then you would have to get into the question of is one day's exposure enough, or two days' exposure, or half a day or an hour, going through a tunnel? You know, it's very, very difficult.

15 So I think my suggestion at the moment, until we have more information, is that those associated with asbestosis in fact are the ones that you have the greatest probability of saying that they are...accurately saying that they are asbestos-attributable.

20 Q. I guess we come back to the question then, how much asbestosis?

20 A. Any amount that a radiologist or a pathologist is willing to say is asbestosis.

Q. On the basis of his or her professional judgement?

A. Yes.

25 Q. Is there any way that one could conduct a study to ascertain medically or biologically, or is there any medical evidence that would link the two? Is there any biological reason that would link the two? Any reason in the cell structure or anything?

30 A. Of course that would be from the standpoint of the sort of analysis that you are inquiring about. That would be desirable, most desirable, if there was something



5 A. (cont'd.) about an asbestos-related tumor that made it identifiable as such. There has been a suggestion from time to time that one particular cell type in adenocarcinoma seemed to occur in greater proportions, somewhat greater proportions, in asbestos workers than in the general population. That's a statistical...and it was a modest increase in situations where this was suggested.

10 But in fact, all kinds of tumors, the other kinds of bronchogenic lung cancers, also occurred in these workers, and if you say that adenocarcinoma is more likely to occur, what happens? Then you say that if he develops a squamous-cell carcinoma that it isn't asbestos-related? I would be no more willing to say that than to say that the adenocarcinoma was.

15 Again referring to my summary comments at Leone, I suggested that it wasn't very helpful at this point in time to direct a lot of energy and investigative resources to the cell-type question, because it certainly isn't going to answer the question in one individual, and I don't think it's going to help us very much to understand asbestos-related carcinogenesis generally, either.

20 Q. So then what you are saying is that there isn't any...correct me if I'm wrong, but is it fair to say that your conclusion is that there may not be any medical or biological way to link the two, but that a fibrogenic dose seems to be about the same as a carcinogenic dose, and therefore it's a reasonable..

25 A. Or maybe lower.

Q. Or maybe lower, and it's therefore a reasonable way to proceed?

30 A. Yes. It's reasonable. I think it deals with probabilities. It will perhaps be inequitable to some people, as most of our systems are, unfortunately. It's the only way that I know to deal with it at this time.





5 Q. Can we just carry on in this article to page 352? You talk in the last paragraph on that page and going over to the next page, I think you talk a little bit about this deactivation process, and I think it indicated even that there is some experimental evidence to suggest that such a process may take place, that the host may deactivate, I guess, the...

A. To some extent.

Q. ...carcinogenicity of the substance?

A. Yes.

10 Q. Can you elaborate a little on that for us?

A. Not really. The citation is from Dr. Cornfeld, who in fact was reviewing the subject in Science several years ago, and simply it is the suggestion that the process of carcinogenesis is not an irreversible process necessarily. That there may be carcinogenic influences that, 15 to a certain level, do not result in a tumor.

Now, if that were to occur...and by the way, let me hasten to say I am not an expert on carcinogenesis, on basic mechanisms of carcinogenesis, but putting it in the terms that I understand it, it's possible that certain repair mechanisms are operative, as they are with other biological 20 processes.

If you and I cut ourselves, certain things happen that ultimately ends up in a scar, which puts the skin back together again, and no harmful effects, long-term harmful effects occur.

25 If we develop pneumococcal pneumonia, we may be very sick but we end up with an essentially normal lung. Nothing, not even a scar necessarily, has occurred after that severe infection. So it isn't totally biologically implausible that the carcinogenic process may also, to a certain extent, have as part of it a repair mechanism, an ability to reverse 30 up to a certain point. And that's all we were suggesting here.



5 Q. Going to the next, the other side of the page, the first full paragraph. You raised the possibility, or raised the fact that others have suggested the possibility that there may be a differing risk in terms of health effects as between mining and milling on the one hand, and manufacturing of asbestos on the other, and there appears implicit in this that the farther away you get from the source, perhaps the greater the risk. Is that a proposition to which you, yourself, in your professional opinion subscribe to?

10 A. Yes. Do you want a simple answer or do you also want a transparency?

Q. A transparency is always helpful, Dr. Weill.

15 A. If you allow me to take a figure from the Simpson report...I suppose everyone here knows about the Committee on Asbestos that was published, a couple of volumes of readable and unreadable stuff...if you look, probably there are a couple of points here. We might even get into a new study that has not been published.

20 I have drawn a couple of things and I'm going to hasten to show you what I have drawn. But what I have not drawn is the following dose-response curves for the respiratory malignancy. This bottom one, which is the flattest one, and that is...by the way, this is really the key figure of the whole report. I mean, it indicates what the risk, lung cancer risk is among the studies that were available at that time...but two I've drawn in, ours and the Charleston one were not available.

25 Okay, the flat one is the McDonald Quebec asbestos miners and millers. These two, this one and this one...

30 Q. Let's just identify those for the record. So the flattest line is McDonald?

A. The flattest one is McDonald.

Q. All right.



5 A. The next one that is part of the actual figure as presented in the publication, and the one after that, are two derived from Dr. Enterline's work. The flatter of the two, the one closest to the Quebec one, are the production workers, and the steeper one are the maintenance workers.

10 Our dose-response curve is right between Enterline's production and maintenance workers, which should come as no surprise because our cohort in fact included both production and maintenance workers - again, an example of what I would consider remarkable comparability among different studies when we all have such doubts about the imprecision of some of our data.

15 Then...so here we are getting further away from the miners and millers, for manufacturing, and then the question of where the end product user lies, no one really knows because for insulators there isn't any good dose-response information, nor has there been any real suggestion. The estimates that we have, or have been suggested to us in terms of average exposures, would put their dose-response curve something like this. But obviously it would be on this side of the manufacturing...on this side, steeper.

20 Q. This is Selikoff's...?

25 A. Insulators, right. He doesn't have individual reconstruction of dose, but when asked what the average exposure of his insulators were, the numbers that he comes would indicate a very high risk indeed for a relatively low level of exposure.

Now, I'm not presenting his data and I'm not going to make a judgement about his data. I'm just saying that if in fact his dose-response curves were available, it would be somewhere over here.

30 Q. Could it even be to the left of the Charleston data?





A. That I don't know.

Q. All right.

5 A. Then we have the second...so I've drawn the New Orleans one, which I felt would be of some help, hopefully, and then I've drawn the later study, which is another study of a manufacturing cohort, asbestos textile manufacturing in Charleston, South Carolina, presented by Dr. Dement and not yet published, in Cardiff. There is a preprint of his paper available. You assume you have it?

10 Q. It's in fact already exhibit four in these proceedings.

15 A. All right. If you draw his dose-response curve for lung cancer, on the same figure, this is what you get. Frightening, obviously. It indicates, I suppose, that one needs now to understand, and this process has begun, why his exposure-response relationships are so different than the others that are displayed on this figure.

20 Basically you have several options. One is that exposure was not appropriately estimated. Another is that response wasn't, that's a little hard to figure, it's a mortality study which generally should be accurate.

I don't know, I have one or two other things that I might just tell you about this study to indicate some possible problems but it may or may not be appropriate to do so.

25 But anyway, getting back to the original question, why I came up, is that what information we do have does suggest an increasing risk from the mine to manufacturing and ultimately, probably, to end products.

Q. Have you got any...

A. Ideas on why this is?

Q. Explanations?

30 A. I don't have any explanations. There are some ideas, I think others have had ideas. One is the



5 A. (cont'd.) possibility that as you go further away from the mine, more and more is done to the fibers. I'm not sure there are any good studies, quite frankly, about fiber dimension and distribution in these various aspects of the industry as you leave mining and milling and go through manufacturing and then to insulation, application, sawing, mixing, tear-out, which of course is another important exposure, whether or not we know what happens to the fibers, whether they get thinner.

10 It seems to me that the more you do to them, and because of the natural qualities of asbestos, it's very likely that they do in fact change in their physical dimensions, to say nothing of what they might absorb on their surface.

15 So I really don't know, but I do believe in conjunction with others, that as you get further from the mine, the risk seems to be higher.

20 There may even be a substantial difference in risk in various aspects of manufacturing. There are asbestos... who knows, it may very well be that the risk in textile manufacturing is larger than it is in asbestos cement manufacturing. The only thing that makes me doubt that is, based on something I've already shown you earlier today, and that is the Roachdale plant is an asbestos textile plant, and the risk of respiratory cancer in a similar portion of the cohort followed long enough and so forth, in their study and our study is remarkably similar.

25 So there are still some questions about that, but I think there is a different risk related to processing.

30 Q. Just dealing with the industry you were studying, can I take you to your summing-up article at the Leone Conference, which is tab twelve. On the second page, page 868. In the second full paragraph you there refer to the fact that some consider that that industry may differ,



Q. (cnt'd.) perhaps in part due to altered surface properties, or possibly because asbestos fibers may be coated with small calcium-containing particles.

5 A. Let me be sure that it's clearly understood what my responsibility in producing this paper was, and that was to summarize the work presented at that meeting and not to give my views...although at times, and I think it's clear where, I do give a view or two.

10 But the point really is that in this instance, this is an example of summarizing, commenting on a view expressed by others. I am not a dust physicist and I can't really respond to the validity of this hypothesis, but it has been suggested more than once in the past that asbestos in asbestos cement does in fact have a coating, and that that coating may make it less pathogenic.

15 There has been some animal work, I think in baboons in South Africa, which tends to support that possibility.

20 I would hasten to say, however, that people in the asbestos cement manufacturing industry are also exposed to pure fiber. I think you can see that in the early phase, in the forming phase of the process before the sheet is made or the shingle or the pipe, or whatever, that sort of argument wouldn't be operative.

25 Q. Is one of the conclusions out of all of this that you should be wary about applying the kind of relative risk assessment that you get in one phase of the industry, to another phase?

I mean, for example, should we be wary about applying the results that McDonald gets out of mining to our manufacturing operations or use operations in Ontario, because we don't have any mines?

30 A. The simple answer to your question, in my opinion, is yes, that I think there are different orders of





A. (cont'd.) risk, as I think I said a few minutes ago, dealing with what phase of the industry one is discussing.

5           You know, there is a good bit of reluctance, and I can understand why, from a compliance standpoint and other things, nonscientific standpoint, in dealing differently with the same substance under different conditions. There is now in our country a precedent for this. I have actually participated as a witness for the Department of Labour, OSHA, in a cotton standard hearing, and the cotton standard in fact now has a different permissible dust level in the early textile processing...carding and opening, and so forth...than in the later processing, such as weaving, and a different standard altogether in nontextile cotton exposures.

10           So I suppose there might come a time, and of course in the U.K. there are different standards for different fiber types in asbestos.

15           Q. Leaving that aside, you really anticipated my next question, which was, would you subscribe to the view that perhaps there should be differing standards depending on what phases of the industry you are talking about?

20           A. In the face of adequate data, yes.

          If you ask me if today, June 16, 1981, do we have enough data to set different standards for different parts of the industry, that's tough. I would say we probably don't.

25           Q. Do you have any other suggestions yourself as to possible explanations for the differences, that I haven't covered with you? I mean the differences between these various studies.

          I take it there are some methodological differences that might account for part of the differing risks that one sees?

30           A. There are methodologic differences. As you



5 A. (cont'd.) know, Dr. Enterline's study dealt with retirees. In some studies there was a far more complete follow of the cohort to death. The Quebec study now has followed their cohort, they have an older cohort, for instance, than we do. They followed their cohort to a very high proportion of deaths. That increases validity, I think.

10 There are some methodologic differences on how you analyse your mortality data. You can use the person-years approach, you can use a case control type of approach. In our instance, they both gave the same answers, but some people prefer one approach over the other.

There are the problems of trace, which will vary from study to study, and we have already discussed some of the problems we had with ours.

15 But I think all in all that the major differences are real differences. I don't think they are methodologic.

Q. There is something that happens to the fiber as it gets farther away from the mine?

A. Maybe.

20 Q. We haven't...I don't think we have all very carefully looked at the Dement paper, but since it seems to have produced some rather dramatic results with respect to, I take it, chrysotile, have you got any helpful comments to us as to perhaps some difficulties with the paper, or what we should be looking for?

25 A. I'll leave you to judge whether they are helpful. I'll try to make them...I will make them brief.

MR. LASKIN: Can we, before we lose track of it, perhaps mark that last projection.

MR. WARREN: The slide from the Simpson report we were talking about.

30 MR. LASKIN: From the Simpson report.

THE WITNESS: A. What I'll do is, I'll see that



A. (cont'd.) you get copies of this. I'll give them to you today. You can photocopy them if you like, right away.

MR. WARREN: Mr. Laskin, should that be number ten?

MR. LASKIN: Are we up to...?

MISS KAHN: We are up to ten.

MR. LASKIN: Okay. It is then number ten?

MISS KAHN: Exhibit number ten.

MR. LASKIN: Thanks, Mr. Warren.

EXHIBIT # 10: The abovementioned slide was then produced and marked.

THE WITNESS: A. One of the problems that Dr. Dement had, and we all have, as we have already discussed earlier, is that in order to generate information that is relevant to present societal, possibly regulatory needs, it would be very nice to be able to correlate adverse health effects with a number or a measurement of asbestos that actually counts asbestos and not all particulates.

So he, as well as some others, have tried to convert, if you will, past measurements of dust, total dust, to fiber. Now he's done that with data that have been supplied to him. The sources have been generally, I think, to company data as well as data from the U.S. Public Health Service. The data go back not indefinitely, not as far back as the cohort goes, as is the case in all these studies, but there is a fair amount of information.

Now, what he did is rather complex. Actually it forms a doctoral thesis, and is available. You can get it from the University of North Carolina, I think. We, in fact, have it. It takes a little while to get it, but you can get it. It's available, and ultimately his paper will be





5 Q. (cont'd.) well, I think the issue of conversion factors from the million particles per cubic feet to fibers is obviously going to be a major issue, and do you think it's possible to set standards based on fibers per cubic centimeter, on studies that were done before that are based on MPPCF?

A. I think there's no alternative. The alternative would be no numerical standard.

10 Q. You did, in fact, in one of your papers, however, quote, results of these studies cannot be used directly in the setting of standards?

A. Directly.

15 Now, I'm not trying to downgrade in any way...and I think my previous testimony throughout the day has suggested that the difficulties in both the reconstruction of exposures using particles and second, in the conversion of those units to units that we now feel is the appropriate measurement for setting standards. I still, however, would maintain that if we don't use this information, what information are we to use?

20 MISS JOLLEY: I think that's all. Thank you.

THE WITNESS: Thank you.

25 DR. DUPRE: Miss Jolley, just before I go to Mr. Warren, and before I lose track of it, part of your line of questioning had to do with asbestotics, and the chairman has a question on observed mortalities among asbestotics, that he always wanted to pose but doesn't dare ask because he is not medically trained...so, Dr. Mustard, could you pose the question I would like to ask?

30 DR. MUSTARD: The question, I think, goes something like this: that individuals with chronic chest disease obviously undergo drug treatment from time to time, and one of the things you so well describe in your articles



DR. MUSTARD: (cont'd.) is that of course you may find in people with chronic chest disease, blood tests which mimic collagen disorders.

THE WITNESS: Yes.

DR. MUSTARD: Therefore, my chairman's question is, how often in practice do you find, or do you ever find, people with chronic chest disease, secondary asbestosis, who show signs of lupus erythematosus...or do you ever see it, and can you relate it back into the problem?

THE WITNESS: For about...well, even longer than we have been studying asbestosis, we have studied a very virulent form of pneumoconiosis produced by sandblasting...sandblaster silicosis...and actually have pointed to this very substantial hazard in shipbuilding and offshore oil industry operations in our part of the country.

In those individuals with very high silica exposures, there is a very high prevalence of antinuclear antibody, up to...Margaret Turner-Warrick has done some of these studies with us, she is the professor of medicine at the Brompton Hospital in London and has an interest in autoimmune disease of the lungs...a very high prevalence, and indeed a very substantial number of those individuals have collagen vascular disorders...a far greater number than one would expect in the general population.

Systemic lupus, rheumatoid arthritis, scleroderma and dermatomyositis.

Having said that, in the asbetotics in our experience, we have found...and Dr. Turner-Warrick has found...increase in a number of antinuclear antibodies and some other evidence of disordered immune reactions in the blood. Some of this work is ongoing right at this moment, and we are correlating the immunologic findings in our unit with the radiographic and lung function findings in the exposure information. But that's ongoing. But suffice it for the



5 A. (cont'd.) moment to say that there are such abnormalities in the blood. We have not to date, however, observed clinically overt collagen vascular disease in asbestotics from this industry.

10 Again, I think it may very well be a matter of degree or extent of the disease. Silicosis in sandblasters is a very aggressive, downhill, rapidly-fatal disease. It's a very, very...it's the most aggressive pneumoconiosis in the world today. Asbestosis, as I've already said, is a slowly progressive disease and it may very well be that that difference in severity and rate of progression explains maybe part of the immunologic findings and the absence of the clinical manifestations that may be associated with those findings.

15 DR. MUSTARD: Let me pose it then, if you did find an individual with chronic chest disease, secondary to asbestosis, and lupus erythematosus, would you become suspicious that there might be a linkage?

20 THE WITNESS: A. Would I become suspicious? Yes. Would I know that there was? Of course not.

Yes, I would.

25 DR. DUPRE: Miss Jolley, did you...?

MISS JOLLEY: Those are my questions.

DR. DUPRE: Mr. Warren, sir?

MR. WARREN: Yes.

30 Mr. Chairman, this won't be very long at all because I'm left with mainly questions here that should be for purposes of clarification.

CROSS-EXAMINATION BY MR. WARREN

35 Q. This morning, Dr. Weill, you showed us a slide which indicated that for both radiological changes, pulmonary changes, asbestosis and lung cancer, with respect to all of these, we were seeing an elbow in the curve at





Q. (cont'd.) essentially a common point in the cumulative exposure. Correct?

A. Yes.

Q. Now, in discussing that chart, you first of all began by making a distinction between the followup period in your study for asbestosis as compared for the followup period for lung cancer. Could you explain to us once again what that distinction was?

A. Yes. The cross-sectional study for evidences of pulmonary fibrosis or asbestosis was a study of a population employed at a certain point in time - 1969/1970. These individuals had on average a length of exposure, and as will be the case in most currently-employed populations, a time since first exposure which was essentially identical since most of these people had continuous service from the time they entered the industry, of seventeen years...which is not bad. On average it's a relatively long-term currently-employed population, and of course, it's why we saw some effects.

The mortality study included all individuals with those requirements, with the least a month in the industry, twenty years, so forth, who had on average twenty-seven or twenty-eight years of followup since first employment.

That means that they had...that cohort had ten more years to develop the effect. Even if the latency period had peaked, there was still an opportunity for more to have that particular outcome of interest.

Since the elbow was in the same place, as you point out, it has been our suggestion that perhaps...and obviously one has to be circumspect about the strength of the evidence..but perhaps the level of exposure with equal followup necessary to produce evidence on x-ray and/or lung function of diffuse pulmonary effects, fibrosis, may be lower than that associated with an increased risk to developing respiratory



A. (cont'd.) malignancy.

5 Q. Now, this morning when we were discussing this precise issue, you mentioned, but didn't elaborate on, support for that proposition derived from Dr. McDonald's study. Could you now elaborate on what support Dr. McDonald's study might provide?

10 A. Of course, this should best come from Dr. McDonald, but it is my understanding that in his latest analysis, published in 1979, I think, in the British Journal of Industrial Medicine, that he had several categories of low-level exposure...I think it was under three hundred-million-particles-per-cubic-foot years...where he demonstrated some excess mortality for nonmalignant respiratory disease, and yet no excess in mortality for malignant respiratory disease. Which again would suggest that the nonmalignant disease was  
15 occurring at lower levels of exposure than the malignant disease, assuming that followup times and so forth were similar.

But the best source for that information is, of course, My friend Corbett McDonald, himself.

20 I may have that paper here, but I'm not sure you want me to work that out.

Q. If this proposition is correct, then it means if we are protecting against an excess incidence of nonmalignant fibrotic disease, that we likewise are protecting against malignant disease, correct?

25 A. That is, of course, the hope and the most useful interpretation if it turns out that it is confirmed, yes.

Q. When Dr. Enterline was with us last Thursday, he suggested...and I think suggested is the best verb to use, and not recommend...he suggested that perhaps in setting standards we should be looking at pulmonary function as our indicator for purposes of standard setting.  
30

Do you have any views on that proposition?



5 A. Well, my first view is that I am absolutely delighted a statistician who has four mortality studies for all of his professional career would have that opinion, and I'll tell him so when I see him next.

10 But seriously, I do feel that lung function is an extremely valuable epidemiologic tool. It's one that I personally, along with the colleagues in my unit, have used intensively for a dozen or more years in doing studies around the countries, using a mobile pulmonary function laboratory. There is a lot to be said about what is necessary when lung function is used in population studies. There must be absolute, almost religious zeal to standardization, for standardization, for optimum technique, frequent calibration of the apparatus, quality control so that curves of spirometric tests are examined frequently, adequate training of technicians. I won't bore you with more. It's a very useful but also very...well, it's testing which isn't so much complex as it is liable to being done poorly, as it is in many places... not only in factories and clinics, but also academic centers.

20 We have several years ago in our country developed standards for spirometry that are now...developed by the American Thoracic Society...and now accepted and used by our government agencies, NIOSH and others, OSHA, so in that sense it's very sensitive.

25 It's also sensitive in following individuals over time, but since the expected changes in lung function over time are so small, and the measurement error substantially greater than the annual change, for instance, very special analytical techniques for constructing individual slopes, and all sorts of other considerations have to be taken into account.

30 Having provided all those words of caution, I would heartily endorse, or hope it isn't too strong to say, embrace my friend Phil Enterline's comments that in fact it





A. (cont'd.) should be a useful way of detecting early effects of adverse inhalant exposures.

5 There is only one other thing that I think is important to say, and I think our data suggested that. For asbestos, I think it is probable that the lung function measurements are slightly less specific than the x-ray. The x-ray isn't specific either. Shadows can occur for a variety of reasons. But if you look at those two things, probably there is more confounding of influences on lung function than there is x-ray.

10 Smoking is an example. As you noted, smoking wasn't a significant determinant of progression radiographically in our cohort, but certainly was a significant determinant of decline in lung function. Undoubtedly there are other effects, 15 allergic respiratory disease, for instance, asthma or other allergic lung problems, and airways problems will have an effect on airways function and pulmonary function, but will not generally influence the chest x-ray.

20 So I think properly used, and proper usage means not only knowing its advantages, but also knowing its limitations, lung function is very useful.

25 Q. Can I interpret that answer to mean that you would favor, given competent persons knowing how to use lung function tests, you would favor using lung function parameters in conjunction with radiological determination, not in lieu of radiological determination?

A. Yes, that's correct.

30 Q. Now, there has been some discussion, considerable discussion today about the criteria which you would use for clinical evaluation in determining whether or not a worker should be removed from continuing exposures to asbestos. Could you summarize for us once again the criteria which you would use in making that determination?



5 A. If in the mind of a competent evaluator an individual who has had workplace exposure to asbestos dust, that individual has...in general this would be radiographic, but occasionally it might be pathologic...usually radiographic evidence of diffuse pulmonary fibrosis at a level that will be selected by the evaluator...and will vary somewhat from person to person by the nature of the beast...in such an individual, further exposure to a fibrogenic dust such as asbestos is, I think, contraindicated because of the evidence that progression ultimately is dose-related, and that even with a latency period, further exposure is adding to that dose.

10 Q. When you use the term diffused pulmonary fibrosis, do you mean to include in that term pleural thickening?

15 A. No.

Q. In making the determination, clinical determination we've just been talking about...that is a determination of diffuse pulmonary fibrosis...would it be appropriate also to consider pulmonary function measurements such as we have been discussing?

20 A. I think it would be appropriate to consider pulmonary function studies in conjunction with the x-ray, but I think in general, to answer the question that you pose, lung function measurements will usually take a secondary role, because of the lower order of specificity that I have already alluded to.

25 Let me tell you when I think it might be helpful. It might be helpful in the individual where, in spite of the competence of the examiner, the x-ray is truly a borderline x-ray...and unfortunately, I hope I'm not disillusioning any of you who don't look at x-rays a lot, but who think that there is something magic about the reading of an x-ray...I hate to say it, there isn't. There are x-rays that are very, very difficult and are truly borderline. They may represent early

30



5 A. (cont'd.) changes of pulmonary fibrosis, a dust-effect associated fibrosis, or they may in fact be on one end of the spectrum of findings or x-rays that you might find in a general population. They are just...that's the way biology is.

10 Now, if in that individual, the questionable x-ray, lung function studies were to show abnormalities consistent with some effect of diffuse pulmonary fibrosis, it might very well help the examiner to decide that yes, there is some effect of the exposure.

15 By the same token, if the lung function measurements in the individual who has truly marginal x-rays is entirely normal, then it might help the examiner to decide...to make his clinical diagnosis in the other direction. That at this time the evidence is not sufficient to say that there is asbestosis.

20 Q. Now, this morning, in response to a question from Mr. Laskin, you explained the reason why you would remove the worker, under these circumstances, to be that although the disease of asbestosis is progressive, once one has made a determination of diffuse pulmonary fibrosis, removal from further exposure lessens the likelihood of further progression, or slowed down the likelihood of further progression. Have I stated that pretty much correctly?

A. Close.

25 Q. Okay. Now, I would take it that although you were discussing this removal in the context of asbestosis, the same sorts of considerations would apply to the possibility of that individual developing lung cancer? That is, further exposure under such a circumstance wouldn't be indicated?

30 A. Yes. I think that's a fair conclusion. Again, we and others have shown that the cancer risks seems to be dose-related. We know, for instance, if somebody already





5 A. (cont'd.) has radiographic evidence of asbestosis, he is no longer on the bottom part of the dose-response curve, he's somewhere else, so that that would be an additional reason for removing him from further exposures.

Q. Now, in the discussion which we have had about pulmonary function, you have noted on several occasions that smoking can cause reduced pulmonary function, as well as asbestos exposure can cause reduced pulmonary function.

A. Yes, indeed.

10 Q. If we have a worker who has demonstrable reduced pulmonary function as a result of smoking, and given the excess incidence of lung cancer for smokers, could significant reductions in pulmonary function be used as a mechanism for taking those heavy smokers out of the continuing exposure, further exposure to asbestos?

15 A. You probably ought to be asking someone else that question. Certainly people who are heavy smokers have increased their risk of lung cancer. People who have been exposed to asbestos have an increased risk as well. The two together have a more than additive, and perhaps  
20 multiplicative or synergistic interaction. Certainly that is something that might very well be indicated.

The practical aspects of whether or not that could be done, of course, are well beyond my expertise or control.

25 Q. I'm not sure I've gotten an answer to my question. I'll pose it in a slightly different way.

From your data there can be no doubt, I take it, that heavy smoking will indeed reduce pulmonary function measured by the tests which we have been discussing here today?

A. Yes.

30 Q. It is possible, is it not, that we may have individuals with severely reduced pulmonary function



Q. (cont'd.) who nonetheless do not show diffuse pulmonary fibrosis by radiological means, for the reasons we have also been discussing?

5 A. Absolutely. As a matter of fact, that probably represents the majority of people who have reduced lung function, in this industry.

10 Q. If we know, as we do know, I believe, that heavy smoking is significantly associated with excess lung cancer...that is, there is an additive or multiplicative effect as a result of exposure to both smoking and asbestos...could the pulmonary function measurement indicating severe lung function deficiency be used as a screening device to remove those heavy smokers from continuing exposure to asbestos?

15 A. Why use lung function as a surrogate to asking whether somebody smokes or not? Implicit in your question is that smoking-related lung function abnormalities indicate some special susceptibility to the development of cancer as well, as opposed to the smoker who doesn't develop...there is no evidence to suggest that, that I know of.

20 Q. What if we did ask the question directly? What about removal of those workers from continuing exposure to asbestos if they are heavy smokers, by any measure that you would like to propose. Is that something that is warranted or in order from the public health standpoint?

25 A. Ask people if they smoke, and if they do, remove them from exposure? If they have a heavy past smoking history?

30 Well, I think I have answered...that was my original answer to the question you didn't ask...or something like that...is that I think since it is a well recognized risk factor, and it is particularly important in association with the dust exposure, that strictly from a public health standpoint one could make a case that the lessening of risk



5 A. (cont'd.) by removing at least one of those risk factors...and hopefully both, one certainly, is beneficial. So as someone interested in prevention, I would say that it makes sense. It's a reasonable approach.

10 Q. If I may ask one more question on this relationship between asbestosis and lung cancer, in response to questions this morning about how one determines whether or not lung cancer is asbestos-related, you indicated that you would hold it was asbestos related whenever there was indication of diffuse pulmonary fibrosis, is that correct?

A. Yes. Asbestosis, right.

15 Q. I think you indicated also that there might be instances in which lung cancer could be attributable in fact, you know, if we knew the truth up there in Plato's sense, where there was not indications of asbestosis?

A. Yes.

20 Q. Could the opposite be likewise true? That is, there could be a situation where there was evidence of asbestosis but where, if we knew the truth in the ultimate sense, that lung cancer was not attributable to asbestos exposure?

A. Absolutely.

25 Q. In reference number seven, I think we discussed on several occasions, at page 352, Mr. Laskin left open one question which wasn't asked.

30 In the last sentence on that page, you say, "It has been recently pointed out that there is no theoretical reason to accept necessarily the linear-at-low-dose assumption, and that there is experimental evidence to support a contrary hypothesis".

Now, in your discussion with Mr. Laskin, you did discuss the first part of the sentence and you referred to the Cornfeld paper. I don't think you elaborated on what you meant





Q. (cont'd.) by that last part of the sentence, that is, "...there is experimental evidence to support a contrary hypothesis"?

A. Cited by Cornfeld.

Q. You did?

A. No. That evidence is in fact cited in the same paper.

Q. That's the evidence you are referring to?

A. That's the evidence I am referring to.

Q. Okay. I don't have many more questions, but let me scratch my head here for a second.

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THE TAPED RECORDINGS OF  
THE INQUIRY PROCEEDINGS

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...to page 141



MR. WARREN: I don't have many more questions, but let me scratch my head here for a second.

5 When Dr. Enterline was with us last week, we had a considerable discussion about this estimates paper which you referred to obliquely this morning, and from your remarks this morning I don't know whether the record is entirely clear on what your position on that paper is.

10 Q. Would you credit the estimate of thirteen to eighteen per cent of cancer in the United States found in that paper being attributable to asbestos?

THE WITNESS: A. No. It's extremely unlikely that the true figure is anywhere near that.

15 Q. Dr. Enterline testified, based on some calculations which he had made, that the true figure was more likely to be in the neighbourhood of one per cent. Is that ---

A. I would say that that's probably reasonable. We have in fact done a specific analysis and we calculated -- made our own estimates on that; but that certainly is far more plausible, based on what information is available.

20 Q. There's been considerable discussion today about the fibre-type distinction between crocidolite and chrysotile, and as I understand the distinction which you made, those distinctions rest upon the epidemiological evidence. Is that true?

25 A. Yes; not just ours, but others which I didn't have time to review, and probably evidence which is familiar to many of you and it will be brought out by some of your other witnesses.

The animal data actually are confusing and do not, in fact -- are not concordant entirely with the epidemiologic evidence.

30 Q. Can you tell us in what sense they are not



Q. (cont'd.) concordant?

5 A. Well, they do not seem universally, or even in general, to suggest a greater risk associated with the amphiboles and with chrysotile.

Q. Would you say that, on this subject of fibre-type distinction, the evidence at this point is not definitive and that it makes sense to keep an open mind?

10 A. Well, like motherhood, I'm always in favour of keeping an open mind.

On the basis of all the information that is available dealing with the mining studies, chrysotile versus amphibole mining, particularly crocidolite, the gas mask information, some of the manufacturing studies, including ours, I think the preponderance of epidemiologic evidence suggests greater hazard associated with amphibole exposures, and particularly crocidolite.

15 I also feel that, where good and particularly consistent epidemiologic evidence (where the evidence is there, it's consistent), it is not in accord with studies performed experimentally in another species; then I would prefer to accept the human studies, because there are inter-species differences.

20 And particularly here I would be concerned about differences that might result from the differing life times, length of life expectancy, in the animal model as opposed to humans, which may relate to the longevity of the fibres in the tissue and some of these other arguments.

25 Now, having said all that, I personally, as a reviewer and evaluator of the literature, and as an investigator with some data of our cells, would agree that the final definitive answers are not there.

30 I would also hasten to say that that is the case in most of what we do, and would again reiterate that I would be





A. (cont'd.) in favour of keeping an open mind.

Q. Good.

5 This morning, when we were talking about the possibility of different risks in different segments of the industry, for purposes simply of clarifying the record once again, you suggested that, as one gets further from the mine, the risks might be greater; and, as I understood that, that was because  
10 you might be dealing with a dust cloud which was more specifically asbestos fibre as compared to other particles which would show up in the particle measurement techniques, or even the fibre measurement techniques.

15 When you said that, though, you meant to say -- you did say -- it was implicit in your statement, I take it, that, when we're talking about the same level of exposure in different sectors away from the mine; correct?

20 A. Yes, indeed. If there are differences -- and this goes for fibre-type differences -- if there are differences by process or differences by fibre type, it is only fair to discuss those differences or infer those differences, if, in fact, dose is kept constant; yes.

25 Q. And, therefore, when there was mention of final products and what hazards might be posed by final products, you weren't in any way intending to suggest the products themselves were hazardous?

A. No.

30 Q. When we're talking about this difference among processes, would it be fair to say that the same phenomenon occurs within any discrete sector of industry? In other words, there are different -- the dust cloud measured in one part of an asbestos cement plant may be different than in another part where different processes are taking place, or different operations taking place?



5 A. That's certainly possible. Exposure is very complex and, unfortunately, it is difficult enough to be convincing, or be convinced, that there are differences among major segments of the industry.

10 As the numbers get smaller and the variation from one plant to another is there, it is obviously much more difficult even to suggest that there may be differences in hazard within a manufacturing installation, for instance; but I couldn't exclude that possibility.

15 Q. My question is not whether we can set a standard which is different for bag opening in the asbestos cement plant as opposed to working with a wet slurry.

20 Instead, my question is whether this phenomenon of differences in potential risk, given the same measurement of exposure, is a phenomenon that occurs, or can occur, not only between segments of the industry but within segments.

25 A. Well, I think I've answer; I said that could be true; we just don't know.

30 Q. From the discussion which we had this morning about the Dement study, am I correct in interpreting the overlay which you put up for us -- I don't know the number, but I think everybody else will recall it -- 10 -- am I correct in interpreting that overlay to say that, if a conversion factor of nine to one was used with respect to the Dement data, that the Dement data would be roughly reconcilable with the field study of the Rochdale plant, which was also an asbestos textile plant?

35 A. Comes close. Whether or not the conference limits would overlap would require some additional analysis and study; but obviously it visually becomes more comparable.

40 MR. LASKIN: Just to keep the record straight, I think it's Exhibit 11, Mr. Warren.

45 MR. WARREN: I'm not going to ask any more



MR. WARREN: (cont'd.) questions about it, so, finally, we have discussed somewhat this morning gastro-intestinal cancer.

5 Q. Am I correct in saying that neither you nor Dr. Peto nor Dr. Dement found any excess incidence of gastrointestinal cancer?

10 A. That's correct; and even further than that, it's my understanding that the Quebec mining study, in the Quebec mining and milling study, there was no overall excess of gastro-intestinal carcinoma either; I think there was some excess in the very heavily exposed, the most heavily exposed, part of that population.

15 Dr. Newhouse's study -- Molly Newhouse's studies originally seem to show a stronger association than her more recent study. As a matter of fact, I think her study presented in Cardiff didn't show an excess G.I. cancer risk at all, so I think more and more there is becoming some very substantial doubt about that finding.

20 Now, this is not to say that the data that did show an excess are not right or were misinterpreted, or anything of that sort; it's very perplexing. There are a number of credible studies that have not found it. Why, I don't know.

25 Q. If we talk about the McDonald study just for a minute on that score, and what you're saying, the data might suggest that gastro-intestinal cancer, if it occurs from asbestos exposure, may be limited to a high-dose phenomenon?

A. Yeah.

30 Q. And I guess it's also fair to say that, from those data, it appears that any excess incidence ceases at levels in excess of levels of where other manifestations of asbestos exposure are occurring?

A. That's the way it appears; yes.

MR. WARREN: That's it for me.





CROSS-EXAMINATION BY MR. STARKMAN:

Weill, cr-ex.

MR. STARKMAN: Yes; I have a few questions. I'll try to be brief. Most things have been sufficiently covered.

Doctor, I wanted to initially ask some questions concerning the data that you used in your studies of the asbestos workers, and I was just wondering, first, were these Johns-Manville plants these people worked at?

THE WITNESS: There were two plants; one was a Johns-Manville plant, and the other was a National Gypsum plant.

Q. Yes. And was the data supplied you by the company, by these companies?

A. Yes; the data was supplied by the companies, but included studies of state and federal agencies.

Q. Well, in what form did you get the data; was it raw data or had it been time-averaged when you received it?

A. In most instances, the data were averaged with the notations of the number of samples going into the averages that we used.

Q. And how many samples would there have been going into an average?

A. I can't tell you how many samples; there were a lot of samples, but I can't give you a number on it.

Q. Did you also get the numbers that went into the averages, just the actual number?

A. Generally, we had a range -- we had a range in addition to the average, yes. Not each individual number, but a -- say, for instance, a particular job had an average of some number, first of all, we knew the number of samples that went into that average, and a range of measurements, a range of values, from which that average was obtained.

Q. But you never looked at the raw data?

A. That's correct.

Q. And do you know who, if you like, synthesized



Q. (cont'd.) the data?

A. Mmm -- in general, yes.

5 I might say that, in the last six or eight years, we've actually made measurements ourselves, and that, of course, went into the paper on -- which was one of these two plants that we discussed earlier today, showing the simultaneous impinger and fibre counting, and so forth.

10 But as far as past data are concerned, the data, as I indicated, either came from the company hygienist or the data from the U.S. Public Health Service that was supplied to the company, and was photocopied and had the man's name on it.

Q. Did you look at the government data separately; did you have a chance to look at that?

15 A. Oh, yes. They're separate, and they were supplied separately.

Q. And for the government data, did you get the raw numbers?

20 A. Well, again, my recollection is that we got the same sort of information that I've just told you; that we'd have averages for a particular work area, with means and ranges for that particular work area.

Q. Now, talking about a specific work area, how often would the testing have gone on?

25 A. I can't tell you that; not very -- by today's standards, it would have been infrequent.

Q. But I mean, just as a rough idea, are we talking hourly, daily, weekly; what ---

A. None of those.

Q. Or just at random?

30 A. There was -- yeah; it would have been, in general, in the fifties and sixties -- would have been yearly or less, in most instances.



5 Q. So, for a particular work area, when you get the average, you might just have -- for a year, you might end up with just -- you get one number a year and, for a decade, you have ten numbers, and then they just average them together; this is the type of thing we're talking about?

10 A. Well, no; I think the averages would have come from more than one number, but, in fact, the sampling at a particular site may have been done only once a year; that's correct.

Q. And, for the plant, how many different sites would there have been?

A. Mmm -- I'd have to look that up; maybe twenty, thirty, forty; somewhere in that range.

15 Q. And then would they average together as well?

15 A. No; they were kept separate, so that we could -- based on the work history and where the man worked, what jobs a man had, we could, in fact, reconstruct an estimate of exposure for that particular site.

20 Q. I think what I'm getting at is, besides showing -- trying to have some discussion as to the credibility of these averages, that if those numbers are particularly high, for some reason -- in other words, because at the time they took it they happened to be measuring a very heavy exposure period during that -- on that day or at that hour; then that would seriously affect the results that we've got, in the sense that the risk would be much greater at lower levels than we're estimating?

25 A. Yes. But I don't know of any reason to suggest or to suppose that that, in fact, was the case. I mean, granted that there wasn't enough sampling, but why there should have been a systematic bias, which is what you're suggesting, 30 over-estimating exposures at that time.





5 Q. I'm just suggesting that -- well, perhaps, because of the infrequency of the sampling, the numbers just may have been higher than an average, in which case we would have seriously under-estimated the risk at low exposures based upon those numbers.

A. Yeah. And if they had been systematically lower, we would have been under-estimating the exposure.

10 Q. Of course. But you have confidence that they're reasonable?

A. I have as much confidence as I think we've expressed publicly or in writing about them; and today, again, I think they are imprecise. I think they're about as good as anyone has. They seem to generate plausible relationships, but I would certainly not give them any more precision than that.

15 Q. Okay.

Working with those numbers, I just want to deal with the death certificates once again, 'cause it is an important matter, I think, and I'm just -- I'm looking at your paper entitled "Clinical problems in asbestos-related diseases," which was written with Dr. Arai. I don't know what year that was.

20 A. Oh, yeah; that was in a book.

Q. That was in the ---

MR. LASKIN: That's in the last part of tab 15; it comes after Dr. Weill's testimony before the Congressional Subcommittee.

25 MR. STARKMAN: Q. Now, I believe here you're talking about mesothelioma, but I'm just looking at the paragraph that begins:

30 "The difficulty in establishing this history is demonstrated by the observation that an average 3.3 medical histories were required before the exposure to asbestos was obtained in a group of



MR. STARKMAN: (cont'd.) patients with mesothelioma, where strong suspicion of asbestos exposure should have existed on the part of the physician."

Would you think that you would find the same problem in relation to asbestosis deaths? I'm sorry; I'm on page 2.

DR. UFFEN: It's the second part.

MR. STARKMAN: Well, it's at the back of the senate -- labour standards testimony.

THE WITNESS: I'm not sure I understand the thrust of the question.

If, in fact, it is that, in the past, particularly in the article that I referred to was written in 1977 -- in the past, that physicians in large and small medical centres -- this happened to be in the Massachusetts General Hospital; they consider themselves a large medical centre, but somebody might not.

Anyway, if, in fact, people were missing asbestos exposures -- exposures -- this is not diagnosis; exposures, histories of exposure -- then I would agree, and that's what we were saying in that paragraph; that people were, in fact, not fully aware of exposures of their patients who had certain conditions that might, or should have led to a higher index of suspicion of such exposure.

Q. Yes; but if they weren't aware of it ---

A. Then they wouldn't diagnose asbestosis.

Q. That's right. So if those numbers held true across, then you'd, in fact, have approximately a third more deaths attributable to asbestos or mesothelioma than in fact were reported?

A. Well, that's a big jump, counsellor. I wouldn't be willing to use the studies of Drs. Kozeny, and so forth, in Boston and extrapolate those to our death certificates; I just -- you know, I guess in the abstract that may be



5 A. (cont'd.) true. I just don't think that we could make that sort of jump; I wouldn't be willing to make that jump.

Q. Well, what number do you think it is?

A. I have no idea. What number do I think the, say, asbestosis might have been under-diagnosed; by what proportion, I have no idea. I suspect it hasn't been by that much.

10 I've been practising in a large medical centre, seeing most of the lung disease related to occupation for about -- well, I hate to admit it, but for about twenty years, and I haven't seen very much in-stage respiratory failure from that disease. I suspect that if there's under-diagnosis, it hasn't been much.

15 And this is in a hospital where one is likely to see most advanced cases -- charity hospital and Veterans' Administration hospital. I certainly wouldn't be willing to say it was under-diagnosed by a factor of three or two, or ...

Q. But there is some factor?

20 A. Oh, I think so -- well, this is true of almost any disorder that requires diagnosis; is that people are going to under-diagnose it at times and over-diagnosis at other times.

Q. While we're looking at this exhibit, I'd just like to refer you a few statements that you made in your evidence, I guess, to the ...

A. House Committee.

25 Q. ... the House Committee. On page 382 ...

A. All right, sir.

Q. ... just in the middle of the first paragraph there:

30 "For practical reasons, however, it is my view that pleural or peritoneal mesotheliomas perhaps confirmed by a panel of pathologists expert in a





5 Q. (cont'd.) different diagnosis of these [I can't read that word] an individual who is occupationally exposed to asbestos or his family should be considered work-related and appropriately compensated without further proof of a causal association."

10 A. I think that's what I have said previously this afternoon; yes, sir.

15 Q. In here, you mention that -- I'm looking now at the bottom of 385 -- and you say that, in your own conceptualization, you think -- you'd be willing to think that there is a linear relationship at low dosage? That some risk would be there.

20 So when we were looking at those graphs, the ones that have an elbow, or the one that has -- the one that looks like an inverted 'S,' you would prefer to think, at least conceptually, that there is a lineal relationship even at low dosage -- doses?

25 A. I would say that, again for the purposes of conservatism, that seems like a reasonable approach. I would leave, again, an open mind on what the dose shape -- dose response shape actually is at those levels; but I would be willing to stick by what I said to the committee.

30 Q. I have a... Following the reference in previous questions on fibre types -- there are fibre lengths -- people were asking about the different types of asbestos, but I'm just -- we don't really have to look at it; but in your summary, I believe, of the symposium, you summarize by saying, in general, there seemed to be agreement among conference participants that carcinogenic potential of asbestos is closely related to fibre length. And I first was wondering, is that your opinion?



A. Yes. It's also the opinion, as I said, of most of the participants ---

5 Q. Yes; could you elaborate a little on what it is and what lengths they were referring to.

A. Well, most of the -- again, now, this is something that has to come from animal work because there's no other way to do that, and most of the studies would suggest that the length is -- that's most hazardous in this regard is over eight to ten microns; at least over five, but probably over ten -- eight to ten in length.

10 Q. Are fibres less than five microns detectable?

A. Yes.

15 Q. I mean, other than on an electron microscope?

A. Yes -- depends on the diameter.

20 Q. That was the second part of the question. I mean, what about the relationship of diameter to the length of a fibre; is there any opinion on that aspect?

A. The fibre dimensions that seem to be most hazardous are the thin, long ones, and whether or not they are visible by optical microscopy depends upon their -- primarily -- their diameter. Of course, to some extent, the length is very, very short, but certainly two, three, or four-micron fibre might be detectable if it isn't too thin. If it's very, very thin, then it wouldn't be.

25 Q. So ---

A. But what -- I'm not sure I understand. Even if it were detectable by E.M., or electron microscopy, investigators doing animal studies have that tool available to them, to characterize their fibre dimensions.

30 Q. Well, I think I was just asking you for purposes of clarification.

On the issue of smoking, I was wondering if you're



5 Q. (cont'd.) aware of any studies -- and I did ask Dr. Enterline this -- of the effect of exposure to asbestos fibres by someone who was a smoker and stopped smoking, during the period of their exposure.

10 A. I think Dr. Selikoff has some information on that, and I'm not prepared to quote it because I'm not sure that it's been published. I think he has some preliminary data indicating a decreasing risk as time elapses following cessation of smoking. You'll have to ask him.

Q. That would be a relative risk; decreasing relative risk?

A. I would assume that's right.

MR. STARKMAN: Those are my questions.

DR. DUPRE: Mr. Ublansky?

15 CROSS-EXAMINATION BY MR. UBLANSKY:

MR. UBLANSKY: The subject of lupus was raised briefly before.

20 Q. Just as a practical matter, can one distinguish fibrotic effects from lupus, or any other pulmonary fibrotic condition from asbestosis?

THE WITNESS: A. Not really, because even if you said that, to make a diagnosis of asbestosis, you need one or more asbestos fibres, that still wouldn't preclude the, well, scenario, or possibility that there's some fibres in the lungs from exposure, and that the fibrosis was due to something else.

25 So the answer to your question is, you cannot distinguish, as far as I know.

30 Q. Again from a practical standpoint, we've been talking quite a bit about lung function tests. Can you be a little bit more specific in terms of numbers. We've been talking in the abstract about impairment, but I wonder -- well, first of all, is there any standardization in this area?





5 Q. (cont'd.) Can one say that eighty per cent of predicted value means something, seventy per cent means something else, and on down the line -- if you follow me?

A. Well, levels of impairment which are used for certain regulatory or compensation purposes vary tremendously.

Q. Yes.

10 A. In our country, unfortunately, they vary from state to state, as most of you know -- I don't know who's American and who's Canadian here, so you'll have to excuse me.

But I don't know, basically, what you're really asking. In fact, our federal system makes a tremendous difference in what level of functional impairment makes somebody disabled.

15 If that individual is a banker and has chronic bronchitis and emphysema, and is applying for Social Security disability payments, benefits, or whether he's a coalminer and he is applying -- same, you know, federal government - and he's applying for black-lung benefits, there's a vastly different standard of impairment required for determination of disability.

20 It sounds perhaps inequitable; it may not sound inequitable to you. It sounds inequitable to me, but that's another matter altogether.

25 So I'm not sure; the answer to your question depends on who's asking. There isn't any magic number that I know that would universally say somebody is impaired or disabled. It's a very difficult thing.

30 It depends, again, on what -- for what purpose and what job is being considered. Disability depends, to me, on the relationship between functional impairment and energy cost of performing that job. It's rather simple; it's a balance between those two.

And somebody with fifty per cent reduction in his



5 A. (cont'd.) FEV-1, if he's a lawyer, maybe he'll be able to continue his work ... [Laughter] But if he's a hardrock miner, he might not; but you get my point.

So I don't know how to answer your question, counsellor; I'm sorry.

Q. All right. Well, let me try a follow-up that perhaps might help.

10 I have often experienced, dealing mainly in compensation cases, a diagnosis of slight asbestosis, for example, based primarily on X-ray changes.

Can you give me a number with respect to pulmonary function that you might associate with that diagnosis?

15 A. No, I can't, because, with minimal changes on the X-ray, you might have no abnormality of lung function, you might have minimal abnormality of lung function, or you might have lung function abnormalities that are greater than expected from the radiographic changes. There's, unfortunately, variability.

20 Q. Okay. Again, from a practical matter, we've been talking mostly about objective measures: X-rays and pulmonary function tests.

25 Perhaps calling more on your experience as a physician in treating victims of asbestos disease, what role do the more subjective symptoms, if you like, play in this? Chest pain, shortness of breath -- although I guess that is also related to the function, but it may not necessarily be directly related; that type of thing. Where does that fit in?

30 A. Well, it's very difficult, because if, in fact, workers have symptoms that are not supported by objective findings of functional impairment or X-ray changes, or cardiovascular status, then I would have to say -- and I think we're often faced with this -- not often, but sometimes faced with this --



5 A. (cont'd.) that we can't find a basis for the respiratory symptoms being presented by the individual worker.

Of course, I don't have to tell you that there are a number of possible reasons for this. One is, maybe we haven't discovered the right pulmonary function test, that measures that individual's impairment, yet. I would think that's a remote possibility, but possible.

10 Another is that the history, as obtained, is not credible, for one reason or another.

Q. Suppose we take that one step farther, beyond detection. Once we've got someone who does exhibit at least signs, objective signs (slight, if you like, X-ray changes), some degree of pulmonary impairment, pulmonary function impairment; the same question.

15 What about the other symptoms; do they play a role at that stage?

20 A. Yeah. I think someone who has objective findings of diffuse lung disease, and who has symptoms that reflect a clinical effect of that disease, those symptoms should be taken into account in making whatever determination it is that you're making.

If that's a compensation determination, well, generally speaking, that would be taken into consideration, I suppose.

25 Q. Obstructive airway disease; I had some discussion about that. Perhaps theoretically, if one were to display an obstructive pattern in terms of your function tests, as well as perhaps the more common restrictive pattern, in the absence of perhaps any other explanation, such as asthma, bronchitis, would you be prepared at that point to concede some connection?

30 A. What's the hypothesis; a non-smoker?





Q. Yeah.

A. Lifelong non-smoker.

Q. Yeah.

A. No allergic respiratory disease, exposed to asbestos and/or other dust for some period of time, who has, primarily on lung function tests, an obstructive airway disease.

Yeah; I'd have to say that there might be a contribution or relationship.

Q. Lastly, silica's been mentioned a number of times. Is there any -- does silica play a role in this mystery? Does it add to, subtract from, or in any way perhaps have any peculiar effects, interacting, if you like, with the asbestos?

A. In our population, as has been pointed out, these workers have been exposed to both asbestos and silica dust. There is evidence on X-ray and pathologically -- which I don't have time to show you, but I have evidence of -- there's evidence on X-ray and pathologically of both consequences; that is, in one biopsy you can see a classical silicotic nodule and in another area you'll see an asbestos body in a diffuse ... [unintelligible] ... suggesting asbestosis.

As far as I can tell, there's no interaction between the two dusts; I think they both produce fibrosis, and they have some peculiarities in terms of their radiographic pattern, and we see both patterns.

What I don't understand in our population is that we can have people from the same part of the plant who presumably have had similar exposures but not necessarily identical, some who present primarily the -- what appears to be the radiographic picture of silicosis and others who seem to have primarily the radiographic consequences of asbestosis.

That may represent some host differences or it may represent exposure differences, but I'm not sure.



MR. UBLANSKY: Thank you, Dr. Weill.

THE WITNESS: You're welcome.

5 DR. DUPRE: Mr. McNamee, before you pose your questions, we might just pause for a moment.

We have, Dr. Weill, something that's known as the Commissioners' hour around here; in other words, the Commissioners get a chance to pose a few questions, and casual, whispered conversations with my two colleagues indicate that we do have some questions to pose.

10 So I will put it to you at this stage whether you would like to break until, let us say, 7:15, and reconvene at that time; would that be all right with you, Mr. McNamee, or would you rather proceed right away?

15 MR. McNAMEE: Well, I thought I'd be just slightly longer than I was the other day, when I asked no questions at all, and I think that maybe five minutes would clear up my questions; maybe then the Commissioners could get together for their questions. I mean, I may as well clean up ---

20 DR. DUPRE: Why don't you go ahead then, Mr. McNamee.

MR. McNAMEE: Yes.

CROSS-EXAMINATION BY MR. McNAMEE:

MR. McNAMEE: Doctor, you said plant A and plant B represented Johns-Manville and National Gypsum, and I was wondering which was which.

25 Q. The plant A was a single [inaudible] and the other produced a mixed variety of product. Which was which?

THE WITNESS: A. B was Johns-Manville.

30 Q. I gather you've now been made aware of this school program in Ontario. Some of the questions today have been directed towards that. Has that background been furnished to you, about the massive program of taking out and encapsulating



Q. (cont'd.) asbestos in schools throughout Ontario?

A. No, it hasn't.

Q. Is there any similar program -- is there a program down in Louisiana about the schools removing asbestos; is there any program?

A. The policy, I think, in our region -- and this is probably true in most parts of the States -- is for an inspection, visual inspection, of installations, material which may contain asbestos, to be completed by the school boards or their representatives.

And in cases where there is damage, disrepair, flaking off of problem material, the repairs to be made. I don't know of any universal or comprehensive program of encapsulating, or doing something to all the material that's in place. Is that what's happening here?

Q. Well, something along that line; I'm not quite sure.

At any rate, Mr. Laskin asked you a question about schoolchildren, which seemed to relate to their susceptibility; and I just note that in one of your studies -- I think it's tab 4, which is headed -- excuse me; I'm getting a bit of a cold -- lung function consequences of dust exposure in asbestos cement manufacturing plants.

This question of susceptibility is indirectly raised; page 90, you talk about ethnic differences and standardization of lung function. And it appears that you have made certain adjustments, because of lung function, between black and white races.

I'm just wondering, is there, if you detect any difference in susceptibility between the two races?

A. Well, no. The differences in lung function





5 A. (cont'd.) between blacks and whites, the expected lung function, which in fact was published separately in another paper which I doubt that is in here, and was also published at the exact same time in another journal by other investigators, and we didn't know they were doing it, and we found very much the same thing.

10 And this is strictly -- it's a size thing; it's just the size of the chest in relation to total stature, which I don't think, in the sense of the word that I think you are implying, really represents a susceptibility factor.

15 It's just that when you're looking at lung function as a way of monitoring respiratory health, and you've got a mixed racial population, you have to account for the differences in lung sizes between the blacks and the whites to put them on an equal basis by which to determine abnormality.

And in order to do that, you have to multiply by a factor that is perhaps less important than just to say that it's not a susceptibility; it's just a racial characteristic dealing with lung size in relation to total height.

20 Q. Well, just to clarify that, for instance, certain women are more susceptible to forms -- certain forms of cancer than men. So what you're saying, that this lung size really doesn't indicate any difference in incidence between the two races; is that correct?

25 A. I don't know of any evidence that the lung size has any influence on such differences -- any such differences that might be found.

30 Q. Because I think there were some, in some of the earlier briefs with respect to schoolchildren, I think some people implied that there might be because of the higher metabolism weight of the children, that there might be greater susceptibility to a disease brought on by exposure to asbestos.



Q. (cont'd.) Do you have anything to say about that?

5 A. I don't -- I don't know of anything that would directly address that question.

Q. Would it be possible that, say, somebody with a higher rate of metabolism than a child, he might ingest more easily, and he also might dispose of the asbestos more easily?

10 A. Inhalation here primarily, rather than ingestion, and metabolism ---

Q. The inhalation might be greater than metabolizing of the same substance?

A. Well, in the usual sense of the word, the body doesn't metabolize asbestos; I'm just not sure ...

15 Q. Dr. Enterline, the other day, indicated that he had done some studies on non-occupational exposure to asbestos and how many additional cases of lung cancer could be caused in a given year, and also how many cases of mesothelioma could be caused.

20 And he indicated that non-occupational exposure of two hundred and forty-five million Americans would cause approximately twenty-eight additional lung cancer deaths per week, per year, and that would be statistically insignificant, would it not?

A. Well, Dr. Enterline is a statistician. Did he say it would be insignificant? It's a small number.

25 Q. Would this twenty-eight -- would that -- I notice that you yourself have indicated that below a certain threshold, that perhaps there is very little excess risk of cancer.

A. In our working population; that's correct.

30 Q. Would you have any, or have you formed any opinion, say, that, to get back to, say, schoolchildren in an environment where the asbestos is fixed and it's not friable and



Q. (cont'd.) loose, whether there'd be any real excess hazard from asbestosis or lung cancer?

5 A. I think, based on what we know, even in low-exposure occupational settings, I would think that the risk, under the circumstances that you describe, would be very, very low; if not non-existent.

Q. You might have something different to say about the people removing the asbestos?

10 A. I might.

Q. I have one final question, and I don't know whether I'm stating this properly, so you can correct me.

Did you not state, more or less, that lung cancer does not occur without some indication of asbestosis?

15 A. No, I didn't say that.

Q. **The** occurrence of mesothelioma, do you regard that it requires a prior indication of asbestosis?

A. Does not. Many people with mesothelioma do not, in fact, have asbestosis; some do.

MR. McNAMEE: Those are my questions; thank you.

20 MR. LASKIN: I just had one reply question I'll clean up, Mr. Chairman. It arose out of a question that Mr. Warren asked on the differing hazards from differing fibre types, and the accumulating evidence that would seem to implicate crocidolite.

RE-EXAMINATION BY MR. LASKIN:

25 Q. Can I ask, you, Dr. Weill, does it then follow, in your professional judgment, that you would aim at setting different standards for different fibres?

A. Yes.

30 Q. And the tentative view that you expressed earlier, at a hundred million particles per cubic foot, is that basically a -- I don't want to -- I'm not trying to pin you down





Q. (cont'd.) to anything, but ...

A. But you will.

5 Q. ... but as I understood your evidence previously, you suggested that that -- below that level, as far as your population was concerned, there really wasn't detectable excess risk.

A. That's what the data, so far, shows.

10 Q. And is that a chrysotile, an essentially chrysotile standard only, or a general standard across fibre types?

A. None of the above. I mean, it isn't a standard at all, nor -- well, first of all, as ---

Q. Let me ---

15 A. --- it applies to -- it applies to this population that has had a mixed-fibre exposure.

As you may remember, if we separate -- wherever the mortality study is; I've got it here somewhere.

Q. Seven.

20 A. Okay. We do separate, on page -- I mean, if you would like to do this -- on page 352, if you look at the SMR's of the no crocidolite exposure group, the attempt here was to get a chrysotile-exposed group, you can see what the levels of exposure are for the varying standardized mortality ratios.

25 That suggests, perhaps, that for chrysotile, if we do the proper sort of multiplying back to getting total particulates, this is now the part of the particulates dealing only with the fibrous content (it gets complicated), we might get some sort of estimate of what a chrysotile standard could be.

30 I'm not prepared to do that today, or probably tomorrow either, because it's complex. The question of what should be the appropriate standard is obviously -- and you understand that quite well.



5 A. (cont'd.) But, yes, I think that, with the information that we have and that I summarized briefly before, I would today feel that it is reasonable to consider the different permissible standard based on the type of asbestos fibre being handled.

10 Q. I take it, then -- one final question; just using your open data, I take it, at the present time, you wouldn't professionally be prepared to convert your levels, expressed in terms of particles, back into fibres?

A. No; I'd be willing to discuss it, and I'd be willing to make some conservative assumptions.

15 For instance, you saw the conversion data today. You wanted to just, without getting any firm conclusions, if you wanted to discuss it in the sort of following context, that would be all right, informally.

20 If we took the most conservative figure that one might want to generate from our data, let's use a conversion ratio of one; that would be conservative, I think. And that gives you a hundred fibre per ml-years, and you do the rest of the division.

MR. LASKIN: Fair enough. Thanks very much, Dr. Weill.

25 DR. DUPRE: Dr. Weill, now you've been extremely generous with us, so may I put us in your hands, so to speak. One option would be to break now and return about 7:30 for maybe three-quarters of an hour, or an hour or so; another option would be to remain until about seven o'clock.

DR. WEILL: I would certainly be willing to go the latter option, if you are willing. If I can stand for a moment, and have some water.

[Some unrelated discussion.]

30 DR. DUPRE: Dr. Uffen, would you like to ...



5 DR. UFFEN: I've just got two things I want to raise with you, and I'm not even sure I know how to put the question properly, and it may be something that you can deal with in a minute, and it may be something where your advice to us would be, you've got to deal with it later on.

10 The first one is in connection with the paper in here that deals with measurement techniques. You and some of your associates had worked on the data absorption dust quantities.

DR. WEILL: Yes, sir.

15 DR. UFFEN: Now, this opens up an area of concern about the future; monitoring the work place, can it be done, how should it be done, et cetera. That's the sort of thing that's on my mind right now.

20 And I guess what I'd like to know from you is, what did you think of that particular instrument; number two, out of that, can we look forward to a day when we can have reliable, continuous monitoring; and do you have any advice to us, for us, about the best directions in which to go?

25 DR. WEILL: Well, Dr. Modd, who is my industrial hygiene colleague and who, in fact, played the major role in the testing of that instrument and the writing of the report, told me, just before I left here, because I noted that it had been chosen as one that was of interest to the Commission, that the bottom line at the moment is that he didn't think it was very good.

30 He said that if you have nothing else, it is an estimate, but it didn't perform very well, as you can see from the paper; but it had to be tested, because people were, in fact, using it, and some were touting it as an important advance.

I'm afraid, beyond that, I can't offer very much.





5 DR. WEILL: (cont'd.) We're looking at other ways of measuring air-borne asbestos; for instance, X-ray defraction -- we have a very highly automated X-ray defraction system that we are at the moment using to try to correlate fibre counts with ---

DR. UFFEN: When you say "automated," you mean it does a bunch of samples quickly, or continuous monitoring?

10 DR. WEILL: It's automated in the sense that you get a very, uh -- fairly quick computer read-out of the peaks, so that it measures -- it does make measurements more quickly than in the past, but it's not by any means so rapid that you could literally do hundreds of samples in a short period of time; but it is an advance.

15 And we're looking at that, not so much now at the moment, because that part could be improved. Right now, we want to see is, how good is it in terms of estimating fibre exposure. As you know, it measures not only fibre, but would also measure any serpentine for chrysotile, for instance, that was in non-fibrous form.

20 So you'd really have to know something about the characteristics of the particles, and then perhaps use this as a way of estimating, in a quantitative way, what the air-borne concentrations were.

25 I'm afraid you really would do better by asking that sort of question to somebody who is in fact in that field, like Dr. Hammad or like Dr. Morton Korn at Johns Hopkins, or someone who has had experience in measurement of air-borne fibre. Vernon Timbrell is another individual, in South Wales, at the MRC Unit, who has a system -- and I don't fully understand it, but it deals with aligning fibres magnetically and then counting them.

30 There are all kinds of automated or modern



5 DR. WEILL: (cont'd.) physical methods for trying to improve the accuracy and the speed by which air-borne asbestos can be quantified; and I think beyond those sort of general comments, it wouldn't be really fair for me to be more specific. I'm a little bit out of my most comfortable area of expertise.

10 DR. UFFEN: It seemed to me more important that we should pursue this, and perhaps you can tell me, in the light of the importance of the conversion factor it seems to be taking on. You remember the nine to one, or one to one; we've got a whole spectrum ---

15 DR. WEILL: That's correct; and that's about the range, too. It's about one to nine to one, and that obviously, which you choose for which process will give you strikingly different dose-response relationships.

20 DR. UFFEN: There's a somewhat related one. In the particular case of the textiles, going farther away from the mining discourse that we had earlier today, I found this a bit surprising, because I would have expected there might be some other kinds of fibrous materials of organic nature, like wool or cotton or ---

25 DR. WEILL: I think there are some; again, I'm not an expert in that, so I'm afraid that ---

30 DR. UFFEN: Well, I'll just put it, anyway, then. Under the microscope might be a devil's job to tell the difference between a curly piece of wool and curly piece of chrysotile.

However, you should be able to tell them apart with some other kind of a detector, say, neutron activation is susceptible to the organic.

35 DR. WEILL: You could use a probe with elemental spectrum analysis, and a scanning electron microscope, and certainly tell apart.



DR. UFFEN: Is this being done?

5 DR. WEILL: Well, yes, it is being done. Fibre identification in tissue, for instance, to tell, in tissue, whether there's a chrysotile fibre or a crocidolite fibre, you can -- that can be done. You have to have pretty fancy equipment to do it, but you can certainly do it; identify the fibre very accurately. And you can certainly tell an organic fibre from a mineral fibre.

10 Those types of techniques tend to have, at the moment, limited practicality in wide application, don't they? It'd be hard to imagine that scanning electron microscopy with the appropriate mineral detection capability could be used in every plant or every work place; I'm afraid it's a research tool, more.

15 DR. UFFEN: My other question is sort of a switch-over to a different area ---

DR. DUPRE: If I can ask you to just hold that one for one moment, because I wanted to follow up on exactly what your area of questioning Dr. Weill.

20 Dr. Weill, when you were, indeed, analyzing some of the implications in the Dement study for us, you pointed out that his conversion factor had been one million parts per cubic foot to three fibres; correct?

DR. WEILL: For most of his job areas, that's correct.

25 DR. DUPRE: Then you suggested that it certainly might not be unreasonable to entertain one to six, or one to nine?

DR. WEILL: Yes.

30 DR. DUPRE: In your paper on the lung function consequences of dust exposure, which is, I guess, your tab number 4, at page 97, if I understand you correctly, at page 97,





5 DR. DUPRE: (cont'd.) the conversion factor that you suggest -- but, of course, this is now, as I understand it, is the asbestos cement industry -- it is one million particles to two fibres.

10 DR. WEILL: That was what we suggested at that time, based on the available information on side-by-side sampling in the literature; since then, we have done our own study of that, which is also included in here.

15 And, as you may remember, when we reviewed those data, I suggested, following questioning by Mr. Laskin, that it may very well be that that conversion should be downgraded somewhat; that ratio should be downgraded somewhat. I wouldn't want to, at this moment, be ---

DR. DUPRE: Put it in what kind of an area?

20 DR. WEILL: Between one and two; perhaps one and a half; maybe even closer to one.

DR. DUPRE: Even one to one?

DR. WEILL: Possibly even one to one.

25 DR. DUPRE: But clearly, at this point, what we are looking at is something that reminds us of the importance of knowing exactly what asbestos-related industry we are looking at ---

DR. WEILL: Yes, sir.

30 DR. DUPRE: --- maybe you're into the one to one to one to two area ---

DR. WEILL: Yes.

35 DR. DUPRE: --- but in, say, textiles, one to three, one to six, one to nine.

DR. WEILL: That's right. Well, you're going to hear some -- when you ask Professor McDonald that question, you're going to hear some incredible ratios.

First of all, he thinks that the conversion --



5 DR. WEILL: (cont'd.) well, he really should speak for himself, but the conversion ratio depends on the concentration of dust, and some of his conversion ratios are quite high; very high.

DR. UFFEN: Fifty?

DR. WEILL: I don't think fifty, but maybe twenty or thirty; I don't remember exactly. You can ask him. Some of them are quite high.

10 DR. DUPRE: You mean, one million particles would be equal to fifty fibres?

DR. WEILL: Some -- you know -- I didn't say fifty. [Laughter.] I don't remember exactly the numbers, but there was somebody ---

MR. LASKIN: Three to seven, I think.

15 DR. WEILL: When he looks at in relation to level of exposure, they're all levels of exposure where the ratios are higher.

But then, I think it's unfair for me to make his judgments for him.

20 DR. DUPRE: But quite clearly, though, you are telling me, as a layman, that there may well be a very important correlation between, on the one hand, the kind of asbestos the industry were looking at and the conversion ratio, on the other?

DR. WEILL: That's right.

25 DR. UFFEN: Well, my other question was, uhm, you're the first expert I've run into who's both conversant with asbestosis and lung cancer and the silicosis question. I don't want to miss the chance to ask the question, which may be trivial; I don't know.

30 It's been put to us that we don't have in the asbestos mines, as yet, in Ontario, although we could have, but we have a lot of other mines (base-metal mines, nickel mines,



5 DR. UFFEN: (cont'd.) copper, and the like) that occur in rocks not at all dissimilar from the ultra-basic rocks in which the asbestos occurs. I forget who it was that suggested that there may be a problem of lung cancers that we don't know about, and if we were to have a set of regulations that had to be implemented, we might discover that we had to implement them where we least expected it at the time of the study.

10 Have I painted the picture for you of what the problem is?

DR. WEILL: I'm not sure exactly. The suggestion is that in some of the mining operations, there may be a risk -- a lung cancer risk ---

15 DR. UFFEN: Due to asbestos fibres and silicosis -- silicates -- and that the number of fibres of asbestos could exceed some regulation established by a regulatory authority, and not have an asbestos mine or a factory within sight.

DR. WEILL: Let me answer that this way, and see if it's responsive to your concern.

20 There is no evidence, credible evidence, that silica exposure, or silicosis, increases the risk for the development of lung cancer.

Does that answer the question yet, or shall I go on?

25 DR. UFFEN: Let's see whether I understand it. Is there any evidence of people getting asbestosis or lung cancer who have worked in other mines, but not necessarily in an asbestos mine or factory?

DR. WEILL: Well, I guess if they worked in a uranium mining operation, that would be a possible contributor to the carcinogenic risk.

30 I suppose, in some mining operations, exposures to arsenic or nickel or -- I'm trying to think of what other





DR. WEILL: (cont'd.) possible carcinogens --  
copper ---

5 DR. UFFEN: Let me put it that -- would it be  
possible for someone to have developed lung cancer from asbes-  
tos fibres ---

DR. WEILL: Yeah.

DR. UFFEN: --- and it was not recognized because  
they were working in a nickel mine?

10 DR. WEILL: Sure, that's possible, if there was an  
asbestos exposure. You mean previously or ---

DR. UFFEN: No -- suppose there's an asbestos exposure  
that nobody suspected because it wasn't labelled asbestos mine;  
it was labelled nickel mine.

15 DR. WEILL: And yet there was a fibrous deposit in  
that mine; is that your point? I guess that's possible.

I just don't know enough about the geology of  
asbestos, in terms of the topographical locations of these vari-  
ous deposits, to know how likely it is that a fibrous serpentine  
would happen to be in a nickel mine. I mean, I just don't know.

20 DR. UFFEN: Well, it's a matter of degree.

Now, the kind of dose-response relationships that  
we've seen recently -- say Dempster's, very steep -- suggest  
that it wouldn't be at all difficult to have a regulation  
intended for asbestos control which would have a decided effect  
on other mines, because what had previously been thought of as  
25 an insignificant amount of asbestos fibre in the waste now  
becomes quite significant.

DR. WEILL: Because of a more rigid standard.

DR. UFFEN: Because of a more rigid standard,  
brought on because it would be controversial to dose response.

30 DR. WEILL: I see. Well, all I can say is that  
certainly it doesn't seem like an improbable sequence of events.



5 DR. WEILL: (cont'd.) It certainly might be that there could be that effect, but I'm not sure how one would deal with that.

DR. UFFEN: I guess what I really want to know is, could there be a problem and nobody knows it because they just didn't know to study the right people?

10 DR. WEILL: Yes. Of course, mortality studies now, in all sorts of industries, are proliferating; there are more. People are studying, for instance, mortality in all phases, or very many phases, of the chemical industry. This is now going on very widely.

15 Mining, various types of mining operations, there have been mortality studies certainly that have been done and reported in the literature for decades, really. They're showing some excess risk and some -- I think there was an article maybe twenty years ago in the New England Journal on metal mining mortality that showed some excess, and nobody knew for sure what it would do to arsenic or -- things that are known carcinogens.

20 DR. UFFEN: I just have a gnawing concern, you see, when I read -- or hear about the death certificates had to be changed when they did an autopsy. What about all the guys that were listed as having died of something else.---

DR. WEILL: And then they didn't change; they didn't know ---

25 DR. UFFEN: Because nobody knew enough to even ask ---

DR. WEILL: It's a problem.

DR. DUPRE: Just on the record, I gather that when you said Dempster a few moments ago, you meant Dement?

30 DR. UFFEN: Did I say Dement -- Dempster; all right. Dement ---

[Laughter.]



MR. LASKIN: The fellow with the steep curve is probably ---

[Laughter.]

DR. DUPRE: Dr. Mustard?

DR. MUSTARD: Mr. Chairman, I have a couple of questions in different sectors. I'd like to go back to our friend the macrophage.

DR. WEILL: Yes, sir.

DR. MUSTARD: In which you showed us what may be part of the underlying basic mechanism, and consider it for a few minutes in the light of two streams of questions; one related to clinical detection of disease, and the other related to cancer.

I think you suggested that the macrophage trying to do battle with the right size and shape of asbestos fibre might be a very important key in terms of what takes place. If I understand it correctly, you implied that the macrophage might well release factors that caused the fibreglass to proliferate and do their thing, much of it; is that correct?

DR. WEILL: Yes.

DR. MUSTARD: Do you know if there are any studies now related to fibreglass involving macrophages which show that the macrophages actually do release factors which stimulate the fibreglass to proliferate?

DR. WEILL: Yes, I think there are such studies.

DR. MUSTARD: So that a stimulated macrophage -- and I wanted to say "stimulated" is a very important factor in terms of the response that occurs in the lungs, and that proliferative response is important as far as the fibrosis.

DR. WEILL: Yes, sir.

DR. MUSTARD: The second part of that story is, I think you also inferred that the macrophage contains enzymes





5 DR. MUSTARD: (cont'd.) within it which can be made available when the macrophage is stimulated enough; they may have something to do with the development of the cancer story.

10 DR. WEILL: No; I didn't say that, but I wouldn't dispute it. I just don't know; I am really not very knowledgeable about the various theories, basic cellular theories of carcinogenesis, I would feel, but I'd be happy to hear your ideas on it.

15 DR. MUSTARD: I was wondering what you knew, and I think there is some evidence that has been posed by some individuals that those enzymes may, under some circumstances, modify the DNA cells -- DNA, of course, as we understand, is very important in determining the properties of cells -- but you're not aware of any studies having been done along that line in relation to the macrophage response to fibres and the development of carcinogens as they might relate to the cells in the lung?

20 DR. WEILL: I'm not aware that they have been done. I suspect that, with the fairly profuse work on cellular responses to fibre, much of it in vitro for cell culture, and so forth, that some of those things may very well have been looked at, finding out if there's relevance to some of your considerations.

25 DR. MUSTARD: Another question on sort of this basic area, I recall that, in the case of silica, the particles are sufficiently small that the macrophage can adjust them; is that correct?

DR. WEILL: That's correct.

30 DR. MUSTARD: And, therefore, one of the differences between silica and the asbestos fibre in this story is the fact that the macrophage, as you so vividly described, has a tough time adjusting some of those fibres?



DR. WEILL: Yeah.

DR. MUSTARD: And I don't know whether that's an explanation for the slight difference in the effect of the two particles or not; but, regardless of that, it seems to me that we then have two problems.

As I understand it, a large proportion of the asbestos fibres probably stay in the lung when they get there; some will be cleared out but a portion stay behind.

DR. WEILL: Yes.

DR. MUSTARD: Has anybody done any trace effects to look at what proportion of asbestos fibres get cleared and what proportion stay behind?

It's been done with some particles but has it been done with asbestos, too?

DR. WEILL: I don't know.

DR. MUSTARD: Well, I think we can safely assume that some of the asbestos fibres stay behind.

DR. WEILL: I think we know they do.

DR. MUSTARD: That means, therefore, that, once you have inhaled asbestos fibres, that presumably the macrophages have to start the tissue reaction taking place, and when two fibreglass is divided and laid down some fibrous tissue, we probably can't detect that clinically. In other words, but there still have been changes in the lungs.

DR. WEILL: Yes.

DR. MUSTARD: Do you have any idea how extensive that process has to become before you can detect it clinically?

DR. WEILL: No.

DR. MUSTARD: But, really, then, what we are doing is, we're defining asbestosis as a thing that can be recognized by us in medicine when the person's dysfunction becomes sufficient that our test systems can detect it; but the process



DR. MUSTARD: (cont'd.) itself has probably been going on from the time the asbestos fibres were inhaled.

5 DR. WEILL: I think that they probably occur within hours of the time the fibre is inhaled.

DR. MUSTARD: This leads me into a very difficult question.

10 In my own field of interest, one of the problems with laboratory tests has been trying to determine their value in actually detecting the underlying problem in vascular disease and coronary artery disease.

15 And, you know, you can go and see your doctor and he'll give you a clean bill of health and you can die of a heart attack two weeks later, because, in essence, unless we do certain things to you, we cannot determine the amount of disease you already have in your coronary arteries.

And I would assume that in this room I could be fairly confident even females have got some degree of narrowing of the coronary arteries already -- [Laughter]

20 One of the problems in medicine is taking a test that you have, and then trying to see, in an objective [inaudible] if you can determine how well the test does predict the underlying process.

25 And I'm not sure how you do it in the pulmonary function field. In venous thrombosis, you can do it now and what you find is that many of your tests are about fifteen to twenty per cent out in being able to predict the underlying process.

Has there been any prospective study done of using pulmonary function tests to predict the degree of underlying disease? And I realize it's a catch-22, because I'm not sure how you'd assess the underlying disease.

30 DR. WEILL: Well, your discussion and questions are obviously extremely important, and, as I'm sure you're





DR. WEILL: (cont'd.) aware, I don't have all these answers. Let me make one or two comments.

First of all, at the stage that you're focusing on now, I suspect that pulmonary function, or anything like the measurement of physiologic changes in the lungs that we have available to us now, or likely to have in the foreseeable future, certainly isn't going to detect, in effect, at the cellular level, that early, after the inhalation of an injurious particle.

What has been done, and what is being done, and I think will continue to gain in interest and perhaps importance, is a look at the cells in the alveolar, or being shed from the alveolar spaces (that is alveolar lavage cells), in various diffuse conditions of the sort that we're discussing.

For instance, the distribution of various types of cells (T-lymphocytes, macrophages, polymorphonuclear leukocytes) in the alveolar lavage ... Let me tell you what alveolar lavage -- I see some perplexed faces.

Well, in fact, what happens is that we can ---

[Laughter]

--- seven o'clock, on whatever day this is.

Now, we can now, just as ... All of you know what a bronchoscope is; it's a tube, a flexible tube now, that has a light at the end, and bends, flexible, and lots of mirrors. It's made by the Japanese, making all those wonderful instruments -- that sort.

And you can actually see down into the air tubes; and, in addition to seeing down, once you get the tube down the trachea, the windpipe, to various portions of the lungs, you can wash out some of the cells that are down there, and there are -- and then count 'em and see what kind of cells they are, and that sort of thing.



5 DR. WEILL: (cont'd.) And you can measure certain proteins, immunoglobulins -- I won't bore you with all the details. But there are certain cells that predominate in certain types of conditions; for instance, in sarcoidosis [phonetic] the T-lymphocytes tend to be very prominent, probably related to activity of the disease.

10 In pulmonary fibrosis, interstitial pulmonary fibrosis of unknown idiopathic pulmonary fibrosis, Hamman-Rich-type problems; the polymorphonuclear leukocytes tend to be prominent. We don't know yet in asbestosis, and that -- in fact, I have a very aggressive immunologist who is trying to get me to agree that some of our patients with asbestosis should have this broncho-alveolar lavage, and I have convinced him that that is going to be hard to accomplish, for various reasons, including the fact that a number of these people are actively working and aren't good candidates for that type of procedure. But there are other reasons that it may be difficult to get a sufficient number of patients for him.

15 But it's possible, in the future, we will have good assays of these cells that we were really looking at here in this electron micrograph to tell us something about the earliest phase of this process, which you imply is important and which I agree is important.

20 I mean, you can take those cells and you can do various things with them; you can probably make certain assays, histochemical assays, perhaps. I don't know all of the various things; I'm not a cellular biologist and I just have a peripheral knowledge about the various techniques.

25 But my point in bringing this up is simply that, if we are going to know when some of these early changes occur, and what those changes are like, it seems to me it's going to be through that kind of analysis system, rather than by measuring  
30



5 DR. WEILL: (cont'd.) air that goes in and out of the lungs and diffusing the passing, and various things of that sort. I just don't think that that's the bait.

Now, the question is, so you can demonstrate that something happens, which, in animals, you can; within hours or so, something starts to happen; a macrophage is recruited into the system, and so forth. So what; how does that help us in solving the public health problem?

10 DR. MUSTARD: I suppose it creates a series of problems. It makes the definition of asbestosis, once you can do that, more complex, because it has the precision which you previously lacked, and, secondly, it makes the diagnosis of asbestosis earlier in the time period, which changes the whole nature of statistical data studies in relation to onset, et cetera.

15 DR. WEILL: Yes, indeed, it does.

DR. MUSTARD: Which would change the whole nature of the graphs we've been looking at.

DR. WEILL: Yes, of course.

20 DR. MUSTARD: Which leads me into another follow-up question from that.

You did allude to this in your discussion with some of the other counsel today, and that is that there seems to be some variation of susceptibility of subjects, and I take it, because you didn't take it any further, that we do not have any good data about genetic susceptibility, in terms of asbestos fibres in the process.

25 DR. WEILL: Appallingly little data. Again, in the Leone conference, there was a small section on host factors, or personal factors, that might influence susceptibility, or explain some of the variability among individuals in terms of response; and the information is very limited. I think I gave it about a

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5 DR. WEILL: (cont'd.) sentence or two in the summation, and it was a sentence expressing, as I remember, disappointment.

10 DR. MUSTARD: Finally, in this line of questioning, before I go to the next part, one of the weaknesses of epidemiology in North America, of course, is that we do not have compulsory post mortems on everybody that dies; therefore, we want to ask a question about what's really going on in the population. You do not get access to the data, so the true incidence of people with asbestos fibres in the lungs is unknown to us, I would think.

15 But what about the Scandinavian countries; the Norwegians, until recently, used to do post mortems on pretty well everybody, but I suspect my suspicions are correct; they probably didn't track asbestos fibres.

20 Do you know if any of those countries that have a high frequency of post mortems have actually tried to track the amount of asbestos fibre in the lungs in relation to pulmonary disease?

25 The WHO has never tried to get them to -- those countries -- to do a detailed study of it.

30 DR. WEILL: No, I'm not aware. Actually, I must say it sounds like it would be interesting to pursue this. I'm not aware that any Scandinavian countries, the post-mortem rate was that high.

35 DR. MUSTARD: The Norwegians, I know, did have more than ninety-five ---

40 DR. WEILL: Did they, really? That's very interesting. It would be a potential source of important information.

45 I do know, for instance, in the United Kingdom, that if an individual is in a scheduled job, an asbestos job,



5 DR. WEILL: (cont'd) and there is a possibility of compensation, a post-mortem examination is required; but, beyond that, I don't know.

Unfortunately, that's still going to be a selected bias, perhaps -- certainly selected population.

DR. MUSTARD: Can I now turn to ---

DR. DUPRE: Did you wish to interject something?

10 MR. WARREN: I was just aware of an investigation in Finland about the types of asbestos, and some of the results in Finland. But the question was asked about the Scandinavian countries.

DR. MUSTARD: Is that a post-mortem study, in which they have a high rate of post mortems on everybody who dies?

15 Could I turn to whatever number seven is called in your tapes, and turn to the issue of -- on page 352; it goes over to page 53 -- on [pages turning] risk assessment, and there was some discussion of that today.

20 I'd like to take the questioning in a slightly different way on this. First of all, I have not -- I think I did read the Cornfeld article when it came through, and it seems to me that was a generalized article on carcinogen risk of all kinds of things that would induce cancer; is that correct?

DR. WEILL: That's right.

25 DR. MUSTARD: Therefore, with asbestos -- well, I certainly have no quarrel with the argument that there could be a special defect in the system; I think maybe we should go through this a bit more.

30 It seems to me, from the current data about carcinogens, there is the evidence that you have chemicals that are direct carcinogens; that is, they do not have to undergo transformation, although there may be detoxification or there may not be,



5 DR. MUSTARD: (contd.) and they can be assessed in a variety of simple assay systems now that provide evidence for that in animal experiments.

10 Then there are other chemicals that can be detoxified but also have to be metabolized before they become carcinogens. And it would seem to me that those would be the most likely ones to have a threshold. Is that a fair assumption? It's that pack of chemicals that, indeed -- that either the chemical itself can be detoxified or it has to undergo a transformation before its effect can be manifested.

15 DR. WEILL: I'm afraid, Dr. Mustard, you're asking again a question that I would not feel comfortable answering, because I just don't consider myself expert in this basic field.

20 DR. MUSTARD: My suspicion is that that would be the case, and which leads me then to asbestos, because it goes back to the asbestos in the macrophage.

25 It would seem to me that, in the sense of what Cornfeld's argument is, it would be relatively unlikely that there is a detoxification story or metabolite story with the asbestos fibre in the macrophage. There may be, but that, in a sense, the application of the Cornfeld reference to the asbestos story has got some limitations on the basis of the arguments that were in Cornfeld's reference.

30 DR. WEILL: I would accept that possibility, yes.

DR. MUSTARD: I think there should be a reservation on that.

DR. WEILL: Yes, indeed.

DR. MUSTARD: Okay; that's the point I wanted to make on that.

Now, if I can turn to -- we're going past our time, Mr. Chairman, but I ...

30 Item 11, table 2; this has been bedevilling me,





5 DR. MUSTARD: (cont'd.) not only with your presentation, but also with the previous presentation of Dr. Enterline's. And I guess I may as well start -- [pages turning] -- it's table 2 -- table 2, page 630 in Exhibit 11.

I would like to preface my questioning with a bit of a dilemma that I faced in another field, to share the same anxieties and concerns, and it's the question of trying to get epidemiological data back from human studies.

10 My good colleagues, like Dr. Sack, et cetera, in my own institution, have trained me that the best data comes, of course, from randomized control trial -- obviously, you can't do a randomized control trial in this kind of field because the ethics of it would be, first of all, indefensible.

15 But even when you do a randomized control trial, carefully designed and guarded by one's colleagues, who are experts in the field, the level of controversy that comes up as a result of those studies, in terms of the suitability of the control groups, even though they are randomized from a population base, leads one to have a high degree of concern about the data in any of these studies and how we interpret it.

20 And, of course, equally as important -- and you emphasized it again this morning -- but even in the randomized control trial we were able to sort of monitor the question of drop-outs and loss of cases, because they're extremely important determinants of what the end results are.

25 And the second sort of lesson is the one that Alvin Feinstein, who's at Yale, has been brainwashing many of us with in my own field, and that is, when you move to the next level of epidemiological study, where you have a group, you try to find a cohort of somebody out there with good cases to match them against, it's got all kinds of problems.

30 And, of course, Dr. Feinstein's done superb



5 DR. MUSTARD: (cont'd.) on estrogens in carcinoma of the uterus, in which he's been standing up against ..... epidemiologists, who claim, indeed, there is an effect; but what he's done is, in effect, control groups have pointed out that, indeed, they are so inappropriate that you can't draw conclusions from them, which then leads to my problem, which I realize is a horrendous one in the field of epidemiological studies and occupational health.

10 You talked about this this morning. The thing that really hits me between the eyes as I look at table 2 is, for the people who are exposed to low fibres (that is, less than two mppcf), the standardized mortality ratio ... [unintelligible].

15 DR. WEILL: We certainly would have been, yes.

DR. MUSTARD: And the digested neoplasm was only twenty-one, and that one astounds me -- well, that's just a freak, because I heard that from the first thousand, but I go to the next thousand, it's thirty-three.

20 And I wonder, therefore, if that may not mean that an effective control group ---

DR. WEILL: It's inappropriate.

25 DR. MUSTARD: --- is inappropriate, and that one should be multiplying those factors, as you suggested, by some factor, and I know I could go to a factor of two or a factor of four, which has a dramatic effect on the standardized mortality ratios for respiratory neoplasms, for example.

30 DR. WEILL: Well, let me make a couple of comments about that. First of all, I agree that the low SMR's overall, and for some of these neoplasms has been troublesome, and has, of course, been the subject of a lot of discussion, both in our unit and elsewhere.

First of all, there are other mortality studies



5 DR. WEILL: (cont'd.) done in Louisiana, using U.S. rates that have come up with very low SMR's. As a matter of fact, it's just purely by chance that one of those was done by Phil Enterline in a chemical company; his SMR's were just as low as these and the overall ones were actually lower.

10 Some people have suggested, for instance, particularly for cancer, that our rates are unusually high, our expected rates are unusually high, and you may, in fact, get some immigration that has resulted in some high rates, because there was a time when there was a paucity of medical -- well, centralized medical care in surrounding southern states, particularly Mississippi and, to a lesser extent, Eastern Texas and Arkansas, which may have produced some of these spurious results.

15 Now, we looked at, though, not just SMR's in relation to U.S. and Louisiana, as we've indicated in the paper, but we also did a case-control analysis that, you know, with the analysis similar to the one that has been done in Quebec, where, in fact, for each lung cancer death, five controls were chosen and a relative risk, or an odds ratio, was generated, based on  
20 the lowest level of exposure; and a dose relationship again did not emerge until the same exposure level: a hundred million particles per cubic foot-years.

25 Now, I'm not saying that all of this absolutely eliminates the problems of low SMR's, and, of course, the worst thing -- what is the -- and we've asked this, ourselves, many times -- what is the worst scenario? The worst scenario is the one, I think, that you've just given: that we've under-estimated the lung cancer deaths; okay?

30 Now, the question is, why would we have under-estimated the lung cancer deaths in the low exposure groups any more than there would have been an under-estimate of the overall





DR. WEILL: (cont'd.) deaths?

5 And my brief this morning was that that's not likely to have occurred; and even if you say, well, let's raise the overall mortality to a hundred, if that's what you'd be happy with, and raise the respiratory malignancy rates by about the same proportion. We still, then, would fail to get elevation of the respiratory cancer risk until the exposure is what we suggest is the beginning of the dose-related excess.

10 I'm afraid that's all we've got; that's the defence. You know, everybody -- I think, ultimately, the new data, perhaps, will provide more information. Whether or not it's convincing is a matter that is going to have to rest with the perceiver, or the beholder. Hopefully, it has some merit.

15 DR. MUSTARD: Is there any reason -- I mean, the other side of the coin is, of course, the control group, as you implied, could be clouded with unknowns, which makes their figures higher.

20 DR. WEILL: Well, we have -- well, I already suggested that there might be; there may be some immigration, and all that concept. So there are possibilities.

25 Unfortunately, regardless of the intensive thought that has been directed; as I say, we've had consultants on this and our own -- well, consultants, critics, whatever you want to call it; everybody has tried taking a shot at this. We've discussed it publicly in meetings, and various things.

30 And I'm afraid we're left with what I have said here today: that we have reason to believe that there hasn't been a -- we don't have misleading results; there are still reservations, almost certainly, that some people will have, until the trace rate is better. And all we can say is that we'll just have to wait.

DR. MUSTARD: Except that if one took the stand --



DR. MUSTARD: (cont'd.) see, the thing, I guess, that really concerns me is, I just can't see why the digestive neoplasm ---

DR. WEILL: I can't tell you; but if you look at (as I'm sure you have) the mortality studies, generally, you're gonna find that the SMR's bounce around; there's going to be a few small ones. A lot of it depends on the number of expected values. They don't all get to be a hundred -- or even ninety. I know some of these are very small.

DR. MUSTARD: It's where the big numbers are in your population group that I see it.

My concern is that, if you had to sort of put a correction factor of two or three for that, because of that effect ---

DR. WEILL: Well, I wouldn't be willing to ---

DR. MUSTARD: I realize that there's no justification for doing that, based on the data, but the uncertainty is there sufficiently, and I'd be interested in what Feinstine might think of this problem, in general, because I think ---

DR. WEILL: Well, maybe he'd be a good one to discuss it with.

DR. MUSTARD: Because it has an enormous bearing on that initial point, in terms of time of malignancy period; that's one of my concerns. And I think that, as a commissioner, I'd like to express my concern about that problem and just re-emphasize what you brought up this morning.

You'd agree that that concern does have an effect on the estimates of latency period, et cetera?

DR. WEILL: It does.

DR. MUSTARD: Thank you.

DR. DUPRE: Dr. Weill, would you just please indulge me very, very briefly. I wanted to make sure I've



5 DR. DUPRE: (cont'd) gotten something straight that you stated this morning, when you actually got down to speaking about possible standards.

Did I understand you correctly to say that a standard that was set at a level that would preclude excess mortality from lung cancer might not be a standard that is low enough to prevent asbestosis?

10 DR. WEILL: Well, I did say it that way, but I think that saying it the other way, that hopefully a standard that would prevent asbestosis would prevent excess lung cancer; but the way you said it is correct as well. Well, I mean it's as correct as what I said.

15 I hope I didn't imply (I didn't mean to imply) that I have all the evidence that would allow that to be an unassailable conclusion. I suggest that that may very well be the fact, and there is some evidence to support it.

20 DR. DUPRE: Now, I fully appreciate what you've said in response to the counsel here, but one cannot use epidemiological studies as a direct basis for standards; they are part of a number of considerations.

25 Is it unfair, though, to ask you this: just in terms of, you know, the kinds of existing or suggested standards that are bouncing around -- two fibres, one fibre, point five -- is there anything to be said about which standard may be, at one and the same time, sufficiently low to prevent asbestosis; and, of course, by implication, preclude excessive deaths from lung cancer as well?

DR. WEILL: Mr. Chairman, I'd like to answer your first question, which was, is it unfair to ask that question. The answer is, it is not unfair to ask that question.

30 As I said before, I really am not prepared to answer it, I'm afraid. I don't have a wealth -- this is --





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DR. WEILL: (cont'd.) having been in the business -- and I don't devote all my time to this; we have a lot of other occupational lung disease research programs going on in my unit, but I do -- I have spent a fair amount of time on the asbestos portion, for about ten to twelve years, and I must say that, in the last year or so, there's probably been as much new material (epidemiologic, Dement, our stuff and our re-doing the mortality), which will give me greater or lesser confidence in what we have now, and the updating of the Quebec material. There's an awful lot of -- Dr. Enterline has had some changes in his interpretation over the years.

So I must say that my present position is one of needing some time to reflect fully on the new information, and try to integrate it in my own mind, trying to decide just where the best judgments lie.

And this afternoon, I think -- I would -- it would be unfair to be as specific as you have every right to ask me to be, but I don't think this is a good time to do it.

DR. DUPRE: I appreciate that; and I appreciate, on behalf of all here, and all my colleagues, even more, your tremendous generosity with your time and your sense of public service for having come all the way up here. Thank you, indeed, very much, Dr. Weill.

DR. WEILL: Thank you; I enjoyed doing it.

[Discussion re resumption.]

INQUIRY ADJOURNED

THE FOREGOING WAS PREPARED  
FROM THE TAPED RECORDINGS  
OF THE INQUIRY PROCEEDINGS

  
DEREK WEST



5 A. (cont'd.) published, but the published paper will almost certainly not have as much in the way of detail of what he did in terms of environmental assessment as the dissertation does.

10 Anyway, he goes through a lot of analytical techniques to generate information concerning exposure estimates in terms of converting from particles to fibers. Now what I'm not going to be able to do is...actually Dr. Hughes in our group has done some of the analyses of what he has done, and come up with some of our own conclusions which differ somewhat in which conversion factors are most appropriate.

15 But to show you how critical it is which conversion factor you use, I have a transparency. That is, I think, perhaps the thing that you might be most interested in.

20 Now, this is from John's own paper. This is from Dr. Dement's paper, and again I want to be sure I want you all to know which lines I have drawn in and which line he has..his lines for lung cancer mortality is this one that I have labelled three to one. He chose...and the six and the nine are mine, but we'll get back to those in a moment...he chose to convert all of the exposures in the plant...well, not all of them. He had some exclusions, but most of the conversions, and I think that includes carding, most of the conversions were in fact three to one. That means for every one million particles per cubic foot, there was three  
25 fibers per ML, as the exposure assignment.

30 Now, just taken by itself, that's a little strange. And when I say strange, it's a little low, because even looking at the work on simultaneous sampling that I referred to before, Eyre and Hurst in the U.S. Public Health Service Hospital, in textile manufacturing, they suggested at least a six to



one ratio, if not higher.

Anyway, that's neither here nor there.

Looking at some of the same data that he had available to him, it also appears to us that the conversion factor of three to one is not appropriate.

Now, you have to remember...three to one, you may say well, that's higher than what we decided is probably true in asbestos cement manufacturing, but we've got all kinds of other dust in that manufacturing situation. Whereas in textile manufacturing, although nothing is absolutely pure, a very much higher proportion, I would expect, and I think this has been confirmed by people who know something about the industry, a much higher proportion would in fact be in asbestos fibers dust.

Q. To put that in some perspective, as I recall from your articles, somewhere between fifteen and twenty-eight percent of your final product had asbestos, and are you then saying that if you look at the final product that the percentage of asbestos in, say textiles, would be much higher?

A. I think so, but again I'm really not one to be absolutely firm about the proportion and relate that to the A-C products. Certainly in textile carding they are using, not exclusively, but primarily, asbestos, as far as I know.

Now...so we've just drawn what would happen if Dr. Dement had decided to use a six-to-one conversion factor, or a nine-to-one, and neither of those estimates are way out of line, by any means. Either of them could be supported, and you can see what it does. It does a lot to the dose-response curve. It brings it down considerably from here to here to here, and in addition to these lines, we have drawn one other thing for you, and that is the only other textile study that I know of that attempts to relate dose





A. (cont'd.) to lung cancer risk, is the Roachdale study, Peto.

Unfortunately, in that study they don't have individual reconstruction. Julian is...is he coming here?

MR. LASKIN: Yes, he is.

THE WITNESS: A. Ask him why he hasn't done that. I say that not entirely facetiously, because in fact a number of years ago that was supposed to be his next project, where they were going to reconstruct individual exposures rather than his saying his entire population had such-and-such an exposure.

But anyway, when you do that and look at what the SMR for lung cancer is in his group of workers who had two hundred fibers per c.c. years...and for some reason also John Dement decided to make things even more difficult for some people, and instead of doing this in fibers per c.c. years, he's calling these thousand fiber per c.c. days down here. I don't know why. But you can convert that by doing really simple mathematics.

Anyway, if you look at where the Roachdale lung cancer SMR is for population exposed to two hundred fibers per c.c. years, and these are people with over thirty years' followup, this is where you are.

So you see, on a nine-to-one conversion ratio the Charleston study isn't really that far away, not knowing where the confidence intervals are here either...really not that far away from the British study.

But if you use this one, the one he used and defends, it is quite a different risk per unit exposure.

Q. The Peto study is in...?

A. (inaudible statement)

Q. I appreciate that, but can you help me on...is the measurement in particles or fibers?



5 A. Fibers. But...well, I'm glad you jogged my memory on that. That's another problem, that fibers measured in Britain at that time are not necessarily equivalent to personal membrane filter samples measured by us today, or yesterday or a week or a year ago, and there may very well be some additional...this is already the result of some manipulation of the number, based on a series of events, of different kinds of counting, area counting versus personal counting and that sort of thing, but it is true that there may be some differences in the methodology of fiber estimation.

10 That's absolutely correct.

Q. What's the steepest curve that you have drawn there?

15 A. I haven't drawn that. I only drew the ones...

Q. Sorry.

A. ...I only drew these two. The steepest curve is a very interesting curve. It's a curve that I have trouble understanding, but let me tell you what it is.

20 It's Dement's curve, and that's the mortality relationship, dose-response relationship for mortality of nonmalignant respiratory diseases. So what he is finding here in his population is a steeper mortality, higher SMR, for nonmalignant respiratory disease...

Q. Asbestosis?

25 A. Most of it, he says, is pneumoconiosis, presumably asbestosis.

Q. That is certainly at odds with the result of your own work?

A. Yes.

30 DR. UFFEN: By way of clarification, I'm getting a bit lost.

THE WITNESS: I can understand that, I'm sorry.



DR. UFFEN: Did he measure particles per something or other, and convert to fibers? Or did he measure fibers?

THE WITNESS: A. He did both.

DR. UFFEN: He measured fibers?

THE WITNESS: A. They measured, in more recent years they did both, in order to generate information on the conversion, and then they went back and used that conversion in converting the old particle counts to fiber counts.

DR. UFFEN: Presumably that is testable, reproducible by an independent...you should be able to check. If you measured both and you do it wrong, if somebody else does it, another lab, and doesn't do it wrong, then you could have an independent check.

THE WITNESS: A. Sure.

DR. UFFEN: Whereas when you go back from trying to use guesstimates, there is no way you can make an independent check?

THE WITNESS: A. Yes. The problem is that in order to know what those exposures in the forties...and most of this cohort, most of these lung cancer deaths come from people who were employed in the forties, I'm quite sure I remember that correct. You have to be able to simulate those conditions, which were wartime conditions, longer, probably, than eight-hour shifts, blackouts I assume, less ventilation because of those constraints on light, and things of that sort. I think it would be very hard to simulate the conditions and make the measurements today.

You could certainly do as we did, and as I showed you earlier, you can certainly do the side-by-side counting there.

DR. UFFEN: Is that being done anywhere else but in textiles?

THE WITNESS: A. Well, in textiles? You mean...





Q. (cont'd.) my time. Sorry, Mr. Chairman, just a couple of more question....

A. Interesting in the one before...and this will be the last comment unless you have some further questions... Dr. Dement, I think, found one mesothelioma, so a very surprisingly low or no mesothelioma risk...and no gastrointestinal cancer excess risk.

Q. Have we exhausted your list of additional photographs?

A. I think so actually, because some of the ones you referred to were in the papers, and you've already gone through them.

Oh, no. The only thing...well, the only other thing, as I have just, from our work, two transparencies dealing with latency period, both for asbestosis and for lung cancer...in our own population.

Q. Could you show them to us, perhaps, quickly and we'll just see what they are?

A. Sure. One of these is in...the mortality one is in the mortality in the paper.

From the cross-sectional study we plotted the prevalence of small irregular opacities of this category of one...one-one or above prevalence in the whole population versus years since first exposure. In other words, when do they first appear. I think it's quite clear they first appear after fifteen years.

And I think that's pretty much consistent with what other people...we have not shown this before, but I got it just because one of your questions on your list had to do with latency.

Q. That's part of the goodies?

A. Yeah, the nonpublished goodies.

MR. LASKIN: Let's make that exhibit twelve.



EXHIBIT # 12: The abovementioned transparency was then produced and marked.

5 THE WITNESS: A. Then this is from our mortality paper, and what it does..let's concentrate for the moment, just at the peak, at where we found the excess risk of lung cancer in the over a hundred million particles per cubic foot years, and the SMR's...and this is now plotted against, shown by categories of years since first, since initial exposure - ten  
10 to fifteen, fifteen to twenty, and so forth.

What you will see is that some excess occurs here, but it's not statistically significant. But a statistically significant increase in relative risk or SMR for lung cancer occurs between twenty-five and thirty years after initial exposure, increases until thirty and thirty-five years,  
15 and then plateaus or doesn't increase any further at over thirty-five years.

A couple of points: This happens to be also in complete agreement, or is in complete agreement, with the peak and then plateauing of risk for lung cancer after thirty  
20 or thirty-five years as the New York/New Jersey insulator studies have shown, the peak is in the same five-year interval, albeit their risk in the insulators was somewhat higher.

But as opposed to the suggestions of some, and this has not been published, but we did look at it very carefully, we could not find a relationship between the  
25 latency period, the lung cancer latency period, this period here of say twenty-five to thirty years, and the level of exposure.

Neither could Dr. Howell from the...Gibson/  
Howell I think is the paper, NIHS. Neither could...I have  
30 these notes...

MR. LASKIN: Q. Do you mean average level of exposure?



5 A. No. Let me...you've heard the comment made that as levels of exposure decrease for asbestos, this will lengthen latency period and we won't see the disease until later, okay?

Q. And at the ultimate, until past your life expectancy?

A. Well, that's the good news. The bad news is that we are still going to keep having the burden of disease ahead of us.

10 As a matter of fact, Doug Little in Quebec, Howell, as I mentioned, and Dr. Hughes in our group, in looking at this absolutely do not find that relationship - that level of exposure, intensity of exposure does not in fact influence this latency period.

15 Who says it does? Well, I think the amosite manufacturing plant studies, I think they have suggested that there is that relationship.

Q. Dr. Enterline...

20 A. Dr. Enterline thinks...I think Enterline thinks there is that relationship. Did you...oh, I shouldn't be asking you questions, but I'm not sure he has that from his own data, does he?

I don't know. I'm not sure he does. I suspect he does not, that he...I think he may just be modelling it or doing one of these funny things that statisticians have a way of doing.

25 But certainly I think the Patterson, New Jersey plant experience suggests that that does, but I just thought for your benefit, since you are interested in latency, we definitely looked at it and couldn't find it, and the other two investigators have also looked for it and also couldn't find it.

30 So there is a latency period, it seems to be a bit longer...it peaks, at least, a little later than for





A. (cont'd.) asbestosis, but it has very little in our studies to...very little influenced by intensity of exposure.

5 MR. LASKIN: Just for the record, I think that slide is actually reproduced at page 349 of your article at tab seven.

THE WITNESS: A. Yes.

MR. LASKIN: So we'll keep the record smaller by not marking that.

10 MR. LASKIN: Q. Dr. Weill, I just have about one or two final questions and they really relate to the interaction between smoking and lung cancer from exposure to asbestos.

15 Do you accept the proposition that the relative risk of lung cancer from exposure to asbestos is similar for smokers and nonsmokers, although the absolute risk is much higher, of course, in smokers?

20 THE WITNESS: A. The data that are available come primarily from, again, the New York/New Jersey American Cancer Society studies. The reason that these data have only recently become available is that it's only through the Cancer Society data bank that we really know what the nonsmoker risk is, and therefore can compare nonsmoking asbestos workers with nonsmoking nonasbestos workers. Again, there is some information on this from Quebec as well.

25 I have no reason to doubt those data, and those data say that the relative risk is in fact the same...that is, the multiple of risk is the same for each smoking group. The absolute number of excess cases is different.

Unfortunately, I do not have that information from my own studies.

30 Q. Do you have any reason to doubt the second proposition, which is that there is a multiplier effect between smoking and asbestos exposure?



A. No reason at all.

MR. LASKIN: I think, Mr. Chairman, I have monopolized Dr. Weill probably far too long, and might I suggest, if you are agreeable, that we give the witness at least a little respite for five minutes, and I can check with my friends...

DR. DUPRE: I think we should do that, counsel.

MR. LASKIN: ...as to how long they may want to go. Do I take it, if everyone is agreeable, that the Commission is prepared to sit this evening?

DR. DUPRE: The Commission is prepared to sit this evening, counsel.

MR. LASKIN: Then we perhaps agree to take a brief recess and I can consult with people...say four o'clock?

DR. DUPRE: Four o'clock.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: It's everyone's preference from down here, and the witness's preference, that we try to go through until six o'clock and see how we go, and...

DR. DUPRE: We'll continue then.

Is there an order of proceeding?

MR. LASKIN: I think they have reached some arrangement.

MR. WARREN: I think Mr. Bazin is going to get up.

DR. DUPRE: Mr. Bazin, please.

CROSS-EXAMINATION BY MR. BAZIN

Q. Dr. Weill, just by way of some clarification, I think counsel for the Commission has done a thorough job in exploring the various aspects of the question you have dealt with.



5 Q. (cont'd.) I would like to ask a few questions on the matter of...I understand first of all that your experience has been basically in the asbestos cement industry, and your studies of it, the various cohorts all come from that industry specifically. Is that correct?

A. That's correct, with the one exception that I mentioned this morning, that we have done a radiographic survey of about six thousand marine engineers - a study which is ongoing and so far no publications have come out of that.

10 Q. I understand also that in this particular industry when we are talking about dust, through the process, these particles of dust are not particles of asbestos dust?

A. That's correct.

15 Q. There will be silica, in cases mica, in other cases talc?

A. Yes.

Q. And other various matters that would be in that dust, is that correct?

A. It's a mixed-dust exposure, that's correct.

20 Q. It's a mixed-dust exposure.

Isn't it true, therefore, that your analysis on pulmonary functions will reflect the interference, if I may use that word, of that dust containing all these particles?

A. I'm not sure I understand the question.

25 If the question is, might some of the other particles other than asbestos be playing a part in the measurement of functional change, the answer is yes.

Q. The answer is yes?

A. Yes.

30 Q. Have you been able to ascertain what influence these other particles may have in terms of the overall results that you gathered insofar as pulmonary functions are affected?

A. Since we found early on that when we compared





5 A. (cont'd.) different levels of x-ray profusion for the small irregular versus the small rounded opacities, that the individuals with small irregular opacities at the same profusion had lower, significantly lower, lung function than the same profusion of small rounded opacities. It has been probable, we have suggested, that the more substantial effect on lung function is that associated with those types of opacities which we think are most associated with asbestos exposure.

10 It does not, however, preclude a nonspecific, perhaps, effect of the other components of the airborne dust on lung function.

Q. It does not preclude those?

A. That's correct.

15 Q. Would it be correct to say that the closer you get to an environment where there are not so many particles in the dust, the better analysis you would have of pulmonary functions?

A. Yes.

Q. I'm not sure if...is my question clear?

20 A. You mean a better correlation with a particular type of dust, certainly. If you are trying to compare asbestos exposure with some effect on lung function, it certainly is clear that the less confounding material you have in the air, the better you are in making that correlation.

Is that the question?

25 Q. Yes. You said it in a very good grammatical way.

A. Thank you.

30 Q. Coming back to the total lung capacity and the various comparisons and ratios that you established in the cohort which you examined, isn't it also true that insofar as this analysis is concerned you would have to know if the person is a smoker or nonsmoker to determine these ratios



Q. (cont'd.) from people working in the asbestos cement industry?

5 A. All of those analyses did in fact take into account the smoking history of the individual, and the effects that are seen there are effects that are not related to smoking.

Q. How did you treat the smoking versus the nonsmoking to determine these statistics? Is it an adjustment based on the total population analysis of their smoking experiment?

10 A. Both an adjustment based on their smoking, and look to see whether or not in each category of exposure the distribution of smoking groups was similar.

15 Actually we have another separate paper on just that subject and I'm sorry that I didn't bring it, and perhaps it wasn't of interest to the Commission, but it's a paper that is entitled..it's in my bibliography...Smoking and Dust Effects on Lung Function, with the question mark of whether or not there is any synergism. We concluded that they were not synergistic, and this is dealt in more detail.

20 But we are convinced that what you see there is not a smoking effect. But what I would agree with you on though, as I think I already have, is that it really is an effect of total dust, and not necessarily just the asbestos. Although we have some evidence to suggest that perhaps the asbestos is the most active component.

25 Q. In your paper on Lung Function Consequences of Dust Exposure, which is tab five, I guess, could I refer you to page ninety-four, in the comment section?

A. Tab five?

Q. No, four. I have had to correlate that myself.

30 MR. LASKIN: It's tab four, I think.

MR. BAZIN: Tab four, yes.



MR. BAZIN: It's tab four, yes. My tab five, your tab four.

MR. BAZIN: Q. Can I refer you to page ninety-four of that document?

THE WITNESS: A. Yes, sir.

Q. The comment four or five sentences down, or just about in the middle of that comment section, "The task has been complicated", and I quote, "by recent evidence indicating that the effects associated with asbestos fiber inhalation probably depends, to a considerable extent, on the type of occupational exposure - mining, milling, etc., etc."

Am I correct to assume from what you have just said that it would also be...it could vary considerably given the type of dust which is related to the type of work being carried out?

A. Sure. I think in my comments before the break, concerning my view, which I share with others, that there are differences in what part of the...between parts of the industry in terms of their effect. Those differences might be due to a number of factors, including the constituents of the dust.

Q. Turning to another subject, on a picture shown this afternoon there was a graph indicating...I think it's exhibit nine...showing the expected time lag before asbestosis appeared. Did I understand that that was the purpose of that slide?

Do you know what I mean?

A. Yes, I do.

Q. Fifteen years...

A. Yes, I know. This one here.

Q. Yes. What conclusion, Dr. Weill, would you





5 Q. (cont'd.) draw from an experiment presently going on in a mine in the Province of Quebec, where three hundred and fifty workers have been exposed for the past...since 1957 to date, and there has been no indication of asbestosis?

A. I would say you should continue to follow that population a little longer.

Q. For asbestosis, how long do you think...and it ties into the latency?

A. Yes.

10 Q. The latency question. For asbestos, how long would you estimate that this cohort should be followed?

A. Well, how many people were in fact in the cohort at the start? Was it a substantial number, or a reasonable number?

15 Q. Three hundred and fifty.

A. Were all there at the beginning?

Q. That's right.

A. And they have been followed since...?

Q. Since 1957.

20 A. 1957, which now is a little over twenty years. I would say that after twenty-five to thirty years the health experience of that cohort, if it continues to be negative, will be extremely reassuring in terms of health effects, of the exposures in that mine.

25 Q. Just a few specific questions, if I may, not in particular order. I would simply like to situate in time some of the slides that you showed this morning, and in particular one slide that had a man with a shovel opening up an asbestos bag which you said came from the Province of Quebec. When was this picture taken, do you know?

A. Yes, I do.

30 Q. When was it?

A. I was there when it was taken, so give me a



A. (cont'd.) moment and I'll give you a year, plus or minus one year, how's that? I would say about 1974.

5 Q. Was there any protective equipment available in the plant where this picture was taken?

A. Yes.

Q. Were these pictures actually taken during the work or were they specifically taken for the purpose of having a slide showing what could be..?

A. They were not posed, counsel.

10 Q. They were not posed?

A. That's correct.

Q. But there was protective equipment in there?

A. Yes. That particular individual neglected to wear it.

15 Q. He was not wearing it?

A. That's right.

Q. On the same slide, there is another picture showing other individuals not wearing any protective equipment?

A. Yes.

Q. That again was not a posed...

20 A. That's correct.

Q. ...picture, it was taken right on the premises?

A. Right on the premises as we walked around the plant.

Q. You are aware that there can't be any high intermittent exposure if protective equipment is worn all the time? When needed?

25 A. Well, there are several parts to that question. Yes. I think that under the theoretical construction of your question, if protective equipment, meaning it's protective, was worn all the time, then there almost by definition would not be any high intermittent exposures. As I am sure you are aware, I would think that there are very few situations,



5 A. (cont'd.) at least in manufacturing, where the use of such equipment can be guaranteed at all times by all people, and that the equipment is always functional and always fits the fact contour properly, and so forth.

Q. Moving on another subject. The question of death certificates?

A. Yes, sir.

10 Q. You mentioned that there is a possibility that what is indicated on the death certificates may be different, depending on the various part of the country these may be filled or completed, and you did mention a few factors that could influence what may or may not be on that death certificate?

A. Yes.

15 Q. Is it your experience that in areas where, for instance, there would be an asbestos mine or an asbestos manufacturing company, that doctors in that vicinity would be sensitized to the question of asbestos?

A. That's certainly possible.

20 Q. And one of the possibilities may well be that there is an overmention of the asbestos factor in the death certificates of people?

A. That's possible.

25 Q. Given also that there may be some Workmen's Compensation available for the family of these people, that also may be a factor which would indicate overmention of the asbestos factor?

A. Yes.

Q. On the question of examinations of workers, you mentioned that as far as you are concerned an annual examination was not sufficient for comparison purposes?

30 A. No, I said it was probably unnecessarily frequent.





Q. Every year?

A. Yes.

5 Q. What did you...did you make a specific suggestion...my notes are not clear?

10 A. I indicated that for the purposes of our longitudinal investigation we have chosen every three years, and even with that time interval changes are slow. I would think that in general that would be satisfactory. Perhaps others would like to do it a little more frequently, two years. Somewhere in that range.

15 Q. Tied into that answer you have a reference to a cost-benefit analysis. Were you referring to a cost-benefit analysis relating to those medical examinations strictly, or to an overall discussion on cost-benefit analysis?

15 A. No, I was relating to the examinations.

Q. To the examinations?

20 A. Yes. Whether or not examining how many hundreds of thousand, or whatever, people fall under the regulations, annually, is in fact productive of adequate benefits for that cost.

25 Q. On the question of the medical diagnosis, which you referred to personally as having to make on workers who had an asbestos exposure history, you mentioned the question of removing some of these people from work, depending of course on your judgement as to what the situation was. Are we talking here about, on your part, about a positive medical diagnosis when you think you would make a recommendation to remove someone from the workplace, or are you referring to the question of protection, greater protection for an individual?

30 A. I'm speaking of removing an individual from further exposure, which is not quite the same as saying removing him from his job, because it all depends on whether



5 A. (cont'd.) or not that job is associated with exposure. So that if you will allow me that distinction, I'm saying removing a person from further exposure if, in the judgement of the evaluating physician, whomever that may be, if he or she is qualified, that individual has pulmonary fibrosis, asbestosis.

Q. Which has been determined on the basis of a positive medical diagnosis?

A. Yes.

MR. BAZIN: That's all. Thank you.

DR. DUPRE: Thank you, M. Bazin.

Next? Miss Jolley?

MISS JOLLEY: All right.

DR. DUPRE: Proceed, please.

15 CROSS-EXAMINATION BY MISS JOLLEY

Q. I just have a few questions to pursue, seeing as John Laskin did such a fine job of pursuing most of the questions.

20 I would like to followup on the discussion of your paper on clinical effects and clinical removal....or basis for clinical decision...

A. Yes. What tab is that, please?

I can find it in my stack, but if you have a number it would be easier.

Q. My tab doesn't relate to...

25 A. You don't have tabs? Okay, I'll find it. Go ahead. I think I'll probably remember most of it.

UNIDENTIFIED SPEAKER: Fourteen, I think.

MISS JOLLEY: Right.

MISS JOLLEY: Q. Some of these questions don't relate exactly to that paper, but they are around that.

30 You discussed this morning the whole issue of



Q. (cont'd.) heart disease and asbestosis, perhaps the diffusing capacity or the gas exchange enhancing heart problems?

5 A. Yes. My comments this morning were directed to questions that I perceived were trying to get at what other diseases might be adversely affected by the presence of asbestosis. My answer, briefly and in general, was that a variety of conditions of the cardiorespiratory system could be adversely affected by acute worsening when the reserve of the lung had already been compromised by lung fibrosis, and I gave as an example pneumonia or heart failure might be tolerated less well by somebody who had a diffuse fibrosing disease of the lungs. I really think that's, probably everything is controversial, but I think that should be, from a medical standpoint, reasonably noncontroversial.

10 Q. We've also had a situation, it could prohibit operations on other diseases, for instance, if the asbestosis was fairly advanced? If you were suffering, for instance, from a cancer in another site that wasn't necessarily related to asbestosis, but it couldn't be operated on because of the lung capacity of an asbestotic?

20 A. I suppose that's a possible scenario, yes.

Q. That was in fact a scenario in Ontario.

A. Yes.

25 Q. I think the thing that I am concerned about is, can it in fact cause the cardiovascular disease, or does it just enhance...I mean I understand the enhancement of a condition, but can it also cause a strain on the heart?

30 A. The answer is yes, that all chronic destructive or fibrosing disease, or obstructive diseases of the lungs, chronic, progressive diseases of the lungs, ultimately, when respiratory failure ensues, produce changes in the pulmonary circulation...and I'll keep this compressed...





Q. Please do.

5 A. ...changes in the pulmonary circulation that in turn do put a strain on the heart, as you suggest, ultimately lead to heart failure. That's a condition called cor pulmonale, which simply means heart disease secondary to lung disease.

So yes, it can happen. But it happens because of respiratory failure. It would mean, obviously, that the asbestosis, if in fact that were the cause, would be very far advanced.

10 Q. I would like to just deal with the diffusing capacity, gas exchange, etc. You said that that was not a particularly sensitive indicator of disability until you have a fairly significant advancement of asbestosis?

15 A. You had it almost right. I said it's not a particularly sensitive indicator of an early adverse effect on the lungs, until the disease was well established and all kinds of other things become abnormal. Yes, that's correct.

Q. So that if a physician is using that to determine disability in terms of compensation, that that would be an inappropriate use?

20 A. Well...

Q. At these low...?

25 A. Oh, at these low levels? Well, again I can only repeat it...it won't pick up a dust effect, in our opinion and I think the data, hopefully, show...we think they show...that they won't pick up an early effect. Now, disability is a whole other matter.

Q. Could I pursue...you raised chronic bronchitis, and I wondered if you could deal with the interaction of asbestosis and obstructive lung disease such as chronic bronchitis?

30 A. Well, that's a very complex and very difficult question. Characteristically, and I would say generally, chronic



5 A. (cont'd.) obstructive lung disease is not  
a consequence of asbestos exposure per se. If an individual  
who has had a long-term exposure to asbestos, or a mixed dust  
including asbestos, and has a negative x-ray, but has  
clinically and functionally important chronic airways obstruction,  
and who is a cigarette smoker, it would be and has been my  
judgement that that clinical condition in that individual has  
not been importantly contributed to by dust exposure. There  
10 may have been some contribution, but it's indistinguishable from  
the obstructive airways disease seen in the general population  
of smokers.

15 Now, that's not quite the same as saying that  
asbestos cannot produce some anatomical changes around the  
airways, and probably also some physiologic function changes  
which lead to some limitation of airflow, and I think we  
and others have some evidence that that in fact does occur,  
and probably occurs pretty early, and that's to be expected  
because the deposition of the particles...bronchiole and  
peribronchiole. But that usually is measurable physiologically  
at modest levels, and is not the same as saying that clinically  
20 important bronchitis and emphysema are the likely results  
of this exposure.

25 That's about as...that's basically about as  
far as I can go. The evidence in the literature is not really  
very good on this. There's some evidence from West Germany  
that there is some increased risk of bronchitis in asbestos  
workers, there is a little evidence from elsewhere that  
pathologically one can see bronchiolitis, perbronchiole  
disease, so probably some effect of the airways is present  
in many of the conditions that we in the past have considered  
interstitial diseases that cause only restriction.

30 But in the face of no evidence of pneumoconiosis,  
I would think functionally or clinically important airways  
obstruction is not likely to be importantly associated with



A. (cont'd.) the dust exposure. At least at the moment we can't say that it is. The evidence isn't there that they are causally associated.

5 Q. In that same paper, when you were dealing with mesothelioma, you made the comment that any individual who was exposed to asbestos and developed mesothelioma...and a family member...should be considered to be work-related?

A. Yes.

10 Q. That has impact for compensation...

A. Yes.

Q. ...for family members as well.

A. Yes.

15 Q. Our board at the present time has criteria that require continuous and repetitive exposure over a certain period of time, and presumably what you are saying there is, if you can show, indicate occupational exposure, that we don't have to have these continuous and repetitive exposures?

20 A. Well, ultimately it's a judgement of what constitutes an adequate or sufficient workplace exposure and what does not. That's a very hard question to answer in the abstract, and I think we'll come down to judgements by compensation boards, by litigators, physicians and so forth.

25 I may have said...maybe not in this paper, I can't remember...perhaps in the Leone Paper, I may have said that in terms of requiring evidence for the diagnosis of mesothelioma and for a work history of mesothelioma, that I would be rigid in my requirements for the former, and relatively lenient for the latter.

30 That's paraphrasing what I said somewhere else, and do you follow what I mean by that? I think you...whenever you have a mesothelioma, because of this potential for confusion and misdiagnosis, I would do the very best I can to get a proper diagnosis. Once that's been established, then I





5 A. (cont'd.) think I would be rather lenient in terms of what I would consider an occupational exposure to asbestos. I can't give you a minimum length of time of exposure or a minimum level of exposure, but I would say that reasonable people would be reasonable about that, and I think it will vary from jurisdiction to jurisdiction.

10 Q. One can discuss the reasonableness of the Board, but anyway, speaking of diagnosis of mesothelioma, you dealt with the under- or overdiagnosis of pleural mesothelioma. Is there a fair misdiagnosis of peritoneal mesothelioma to pancreatic cancer?

15 A. I think so. I would be not really the perfect person to go into chapter and verse on that, because I don't do pathology myself and would have to rely on larger series of pathologic material to be sure. But I would say that that is highly probable, that some mesotheliomas are being missed and perhaps at times being diagnosed as metastatic tumor to the peritoneum, and pancreas, as you suggest, would be a suitable candidate for a primary in that instance.

20 Q. I want to ask you a question about...are you familiar with the term 'dust effects'? Or 'preasbestotic' or 'presillicotic' conditions? Do you know those terms?

25 A. I use the term dust effect all the time, but in general when I use it, I am actually referring to an effect, number one, that I think is attributable, and number two, that can be measured.

30 I don't really use it in that sense, that it is a pre anything. It may be an early dust effect, the x-ray changes may be minimal, the lung function changes slight, if there is pathology, histologic abnormalities not striking. Beyond that I'm not sure what you mean.

Q. Would dust effects be a category for removal?



A. Well, if the dust effects that whoever is using that term refers to pulmonary fibrosis, the answer is yes.

5 Q. I would like to ask you a question that came up and I noticed my colleagues from Quebec looked startled when you said it, but you talked about the silica in mining. Do you have any indication...

A. Can I retract that?

Q. Maybe I shouldn't ask the question.

I just wondered why you talked about that?

10 A. No, I don't have any specific information, except that it's recognized that mining...when we were talking about mixed dust exposures as opposed to pure dust exposures, one must consider that in mining of varying types...hard metal and other mining...there is plenty of silica in the earth's crust, and from time to time there will be...and particularly  
15 in past years...there might well have been exposures to chrysolite and silica.

I didn't see them look startled, but maybe I wasn't looking in the right direction.

20 Q. Again you, as did Dr. Enterline, talked about intermittent high exposures and potential hazard involved in that...and again I raise the question because it's happening right probably today...is that we have involved in Ontario, as in the U.S., we've got asbestos removal programs in our schools, etc., and our brothers and sisters in the building trades are ripping out asbestos right now, and some of them  
25 without protection. I guess what I'm asking is, shouldn't we be moving to put rigid protection on that kind of operation immediately...if in fact the short, intermittent high exposures are right?

30 A. Yes. I don't know exactly to what extent and how general full protection of construction workers who are in the process of ripping out old asbestos insulation that has



5 A. (cont'd.) been in place for many years...how general or adequate that protection is. It has been my impression that in general there is increasing protection of that sort being used. Certainly I have seen pictures of individuals who have self-contained air supplies and hoods and that sort of thing...almost totally isolated from their immediate environment.

How widespread the use of that kind of protection is, I don't know. If you are asking do I think it's a good idea to have it, the answer is yes.

10 Q. I would like to deal briefly with smoking again, and that is that in the discussion earlier we dealt with some of Dr. Enterline's material.

15 Dr. Enterline, last Thursday, discussed the possibility of the same thing happening for asbestos workers as happened with uranium...in the uranium mining...and that is the possibility that smoking in fact, what smoking does is shorten latency and ultimately the smoking will become irrelevant, that there will be just as much lung cancer from asbestos. This was a proposition he presented to this group last Thursday.

20 A. He told you that last Thursday?

Q. Yes.

25 A. I don't know what evidence there is for that. I must say that I haven't given it much thought. This is the first I've heard of that. Nor am I aware that the evidence is consistent with that notion in uranium mining....the suggestion being that smoking ultimately becomes irrelevant and influences latency period. I just don't know what evidence there is for that.

If there is, I would like to see it and review it.

30 Q. Well then, proceeding with smoking to the ultimate aim, you discussed in the paper I referred to earlier, you dealt with, "one can clearly disregard smoking in attempting





Q. (cont'd.) to determine the role of asbestos exposure in the individual case".

Again, I would like to pursue the question with you. Does that mean in terms of compensating lung cancer...

A. I'm sorry. Are you...

Q. I'm sorry. On page 383, and again in the Basis for Clinical Decision Making.

A. Yes.

Q. You were discussing Dr. Enterline's material, which was essentially Dr. Selikoff's material, and then...

A. Right.

Q. ...at the very top of the second line it says, "One can clearly disregard smoking in attempting to determine the role of asbestos exposure in individual cases".

A. Yes.

Q. Would it be fair to say that a compensation board should therefore not consider smoking when they are considering lung cancer, with an asbestos worker, for compensating?

A. Well, I think again in individual cases, as I said then, it would be very difficult to know how to use smoking. If the risk is elevated to whatever extent in both smokers and nonsmokers, and one would have to say, if you believe the data, accept the data, that that is in fact the case, then I would say that the smoker or the nonsmoker who develops a tumor and who has had sufficient exposure may have an asbestos-attributable tumor.

As you know, we've gone a little further than that today, and I've tried to indicate, with all it's imperfections, the only way that I know at the moment to make a judgement about the probability of sufficient exposure.

Q. Again, I would just like to ask you about...







